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THE RELATIVE TOXICITY AND EFFICIENCY OF CHLOROFORM, ETHER AND NITROUS OXID-OXYGEN ANESTHESIA IN PREGNANCY AND LABOR.*

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CHICAGO, ILL.

Surgeons and anesthetists are pretty well agreed that the anesthetic should be adapted to the operation and the particular needs of the patient. Naturally there is a marked difference of opinion regarding the choice since we are all influenced by our training and the favorable or unfavorable results obtained in individual cases. Statistics would indicate that chloroform is the most dangerous and nitrous oxid-oxygen the safest anesthetic, but one recent writer maintains that nitrous oxid-oxygen is the most dangerous anesthetic, while another states that when given with pure oxygen chloroform is one of the safest. The writer, with many others, has assumed that nitrous oxid-oxygen was the safest anesthetic to use during pregnancy and labor. In his experience there have been no ill effects which could be attributed to the use of the nitrous oxid-oxygen, yet physicians have reported fatalities and other complications which seemed to have resulted from its administration during pregnancy or labor. The reason for this study is therefore evident.

Fig. 1. Extensive central necrosis of liver. Guinea-pig died 22 hours after 6 hours of light chloroform anesthesia. Photomicrograph x62.

Fig. 2. Marked congestion of the liver of a foetus still-born, after mother had two hours of light chloroform anesthesia. Photomicrograph x62.

EXPERIMENTAL DETAILS.

The literature shows that a comparison of the former experimental studies of chloroform, ether and nitrous oxid-oxygen is open to much criticism since the anesthetics were administered under very different conditions; without sufficient controls; with the animals in unnatural positions, thereby being subjected to changes from shock; and the quantity of anesthetic used was often excessive for the body weight of the animal. Therefore the writer constructed an anesthetic chamber in which groups of pregnant and non-pregnant animals could be anesthetised under very similar conditions. Each anesthetic was administered in a vaporous or gaseous state. The concentration of carbon dioxid was limited by having a false floor three inches from the bottom of the box under which was placed a solution of lime water or sodium hydrate, and having a ventilating valve near the bottom as well as in the top. The animals had perfect freedom and their condition could be observed at all times through the glass door.

The results of this study were reported in considerable detail to the Chicago Gynecological Society, May 18, 1917.

REVIEW OF MAIN GROUP EXPERIMENTS.

I. CHLOROFORM—AIR.

Three pregnant and one non-pregnant guinea-pigs were anesthetised two hours daily until they had six hours of light chloroform anesthesia. After the first anesthesia one pig aborted and ate her young. After the third period a second pig aborted three slightly premature young, and died in convulsions twenty-two hours after the end of the

* Read during the Fifth Annual Meeting of the American Association of Anesthetists, New York City, June 2, 1917. From the Department of Obstetrics & Gynecology, Rush Medical College, in Affiliation with the University of Chicago and the Presbyterian Hospital.

anesthetic. The non-pregnant pig died in convulsions twenty-one hours after the end of the anesthetic. Both adult pigs showed typical central necrosis of the liver, marked congestion of the adrenals and edema of the kidney epithelium. The livers of all three young showed a marked passive congestion, cell destruction and fatty changes. The two surviving adults and the one young guinea-pig born alive subsequent to this anesthesia, had a practically normal appearance of tissue when killed five weeks later.

II. ETHER—AIR.

Three pregnant and one non-pregnant guinea-pigs were anesthetised two hours daily for two days and three hours and fifteen minutes the third period. After the first anesthetic one gave birth to two live young which were anesthetised with their mother the remaining five hours and fifteen minutes. The non-pregnant pig, one of the young, and a normal control animal were anesthetised and killed by decapitation forty-eight hours after the last anesthesia. The livers of these animals showed some loss of staining power and presented a picture resembling that seen after ordinary asphyxiation. This is thought to be a moderate degree of parenchymatous degeneration and tissue swelling. The young pig had evidence of some fatty changes and cell edema. Its mate killed twenty days later had an apparently normal liver. The second ether pig aborted and ate her young. The third pregnant pig delivered two live young nineteen days after the completion of the anesthetic but these died within forty-eight and seventy-two hours after birth. The livers of the etherized pigs were found to be apparently normal twenty-two days after the anesthesia. The two young which died shortly after birth showed loss in staining power of the liver cells and rather marked fatty changes.

The liver changes found after ether anesthesia are of a different type and less severe than those observed following chloroform.

III. NITROUS OXID-OXYGEN ANESTHESIA.

Three pregnant and two non-pregnant guinea-pigs were anesthetised daily for four days until they had a total of seven hours and forty-five minutes of nitrous oxid-oxygen anesthesia. At the end of this period one of the pigs died under an anesthesia from which the others recovered within two minutes after being removed from the box. The sections of its liver showed a pathological condition as evidenced by a marked round cell infiltration. Another pig died of tuberculosis and peritonitis twenty-two days later. It contained macerated young. A third pig which aborted died of pneumonia thirty-two days after the anesthesia. In this experiment all of the young apparently died in utero. The non-pregnant pigs were killed with nitrous oxid asphyxia two months after the anesthesia. The marked peritoneal hemorrhage from the liver of one, and the evidence of bloody serum on the capsule of the liver of both suggests that they had not completely recovered from its effects. A control pig killed at the same time had a marked passive congestion of the liver but no macroscopic hemorrhage from its capsule.

Fig. 3. Liver of young guinea-pig born alive 16 days after 1 hour of light chloroform anesthesia, given with an excess of pure oxygen. Note fatty vacuoles. Photomicrograph x62.

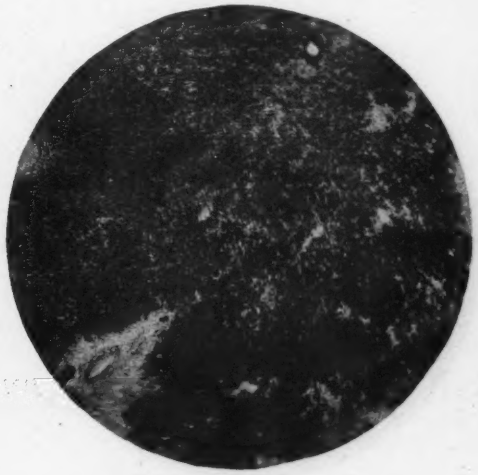


Fig. 4. Sudan III stain of frozen section of tissue shown in Fig. 3. Photomicrograph x62.

IV. NITROUS OXID-OXYGEN ANALGESIA.

Three pregnant and one non-pregnant guinea-pigs were placed in the box and given a mixture containing from 20 to 30 per cent. oxygen two

hours daily for three days. One of the pigs delivered one live young just before the second period and delivered the second while in the box. This young pig lived its first half hour in the mixture. Before the third period a second pig delivered three live young. The four adults and five young pigs were placed in the box for the third period. The remaining pregnant pig aborted the night following the third analgesia. The pig born in the box was found dead the morning after the last period. Twenty-four hours later two more of the young were found dead. Two days later one of the mothers died of peritonitis and forty-eight hours after her death her remaining young died of pneumonia. The still-born young had a marked round cell infiltration of the liver. The pigs dying forty-eight hours after the completion of the analgesia had a passive congestion of the liver, and moderate fatty changes. Those living six days had normal appearing liver tissue. The livers of the adult pigs seemed to be very little affected by the analgesia and appeared normal when killed.

Fig. 5. Liver of normal guinea-pig asphyxiated in bell-jar. Note evidence of congestion and loss in staining power of cells. Photomicrograph x62.

V. NITROUS OXID-OXYGEN ANESTHESIA, ONE HOUR.

Two pregnant and one non-pregnant guinea-pigs were given one hour deep nitrous oxid-oxygen anesthesia. The non-pregnant pig was killed by decapitation fifteen minutes after the completion of the anesthesia. Its tissues appeared perfectly normal. One of the pregnant pigs delivered four still born young seven days after the anesthesia. The other delivered three premature live and two still-born young thirteen days after the anesthesia. The three young died within a few hours after birth and their livers show the round cell infiltration previously observed in still-born animals. The tissues of the mothers appear normal.

It should be noted that with guinea pigs the larger the litter the smaller the young and the greater the mortality following birth.

VI. CHLOROFORM—OXYGEN, ONE HOUR.

Two pregnant and one non-pregnant guinea-pigs were lightly anesthetised with chloroform, forty gallons of oxygen being passed into the box during the hour of anesthesia. The non-pregnant pig was killed forty-eight hours after the completion of the anesthesia and was found to have a liver which is microscopically normal. The pregnant pigs delivered one and two young respectively sixteen days later. These were killed with their mothers by decapitation forty-eight hours after birth. One of the mothers had yellow areas on the surface of the liver which were found to be fatty. All three of the young had very yellow livers and the sections show that they had a large amount of fat. This is seen in the Sudan III stains of frozen sections.

Fig. 6. Liver of guinea-pig killed by decapitation 24 days after 7 hours and 15 minutes of etner anesthesia. Photomicrograph x62.

Various controls were made by killing normal animals with nitrous oxid asphyxia, ordinary asphyxia, etherizing and killing by decapitation, holding and decapitating, asphyxiating with nitrous oxid until respiration had stopped then removing and resuscitating. Sections of liver, lung, heart, adrenals, kidneys and spleen were made from fifty animals. Evidence of hemorrhage from the liver was found after both chloroform and nitrous oxid. None was observed after ether anesthesia.

DISCUSSION.

In the present paper the writer wishes to call attention to a number of points reported in the litera-

ture which support the conclusions which he has reached from his experimental and clinical study of chloroform, ether and nitrous oxid-oxygen. He realizes that his results are not conclusive but offers them with the hope that they will stimulate further and more careful studies both clinical and experimental.

The work of Graham, Sansum, Woodyatt and others indicates that "Chloroform is prone to cause swelling of cells, with fat infiltration, necrosis, and a hemorrhagic tendency. Ether has not been observed to cause necrosis, but it may produce milder forms of parenchymatous degeneration and tissue swelling. Nitrous oxid has little tendency to produce any visible tissue changes. The tendency of chloroform, ether, and nitrous oxid to produce suppressions of glucose and nitrogen excretion on phlorizinized dogs parallels their ability to produce

Fig. 7. Liver of normal guinea-pig killed by nitrous oxid asphyxiation. Tissue very similar to that of Fig. 5. Photomicrograph x62.

Fig. 8. Note the marked engorgement of liver in guinea-pig killed by nitrous oxid asphyxia 2 months after 7 hours and 45 minutes of nitrous oxid-oxygen anesthesia. Compare with Figs. 5 and 7. Photomicrograph x62.

changes in which tissue swelling is an important feature. Working with chloroform it is difficult to narcotize phlorizinized dogs for more than 8 to 10 minutes without killing them; although with ether, narcosis was successfully prolonged for two hours." (Sansum and Woodyatt).

It has long been recognized that a glycosuria may follow a deep anesthesia. The experiments of King, Chaffee, Anderson, Redelings and others show that when given to healthy animals in sufficient doses, narcotic drugs cause a hyperglycemia with or without a glycosuria, that the rise in blood sugar is at the expense of glycogen, and that this is caused by cell asphyxia. Bradner and Reimann found acetonuria in 61.7 per cent. of Deaver's postoperative cases.

Cases of late chloroform poisoning give clinical confirmation to the laboratory production of the central liver necrosis following the administration of chloroform to healthy animals. The present experiments offer some evidence to disprove Gwathmey's belief that chloroform vapor is made safe by administering it with pure oxygen.

Apart from the possibility of the late poisoning, chloroform may be the exciting cause of death in course of an anesthesia. Levy from his valuable clinical and experimental study states that; "Death from ventricular fibrilations under chloroform may be observed under any of the following and allied conditions.

A. During the induction and early stages of the administration of chloroform and exceptionally late in the administration: (1) during struggling and excitement; (2) on removal of the chloroform; (3) on abrupt administration of the chloroform after removal or its sudden increase during a period of very light anesthesia, and (4) by any combination of these occurrences.

B. During operation. By strong sensory stimuli under light anesthesia.

C. After operation. On removal of the chloroform, especially after a short operation.

There can be no doubt that all anesthetics cause changes in the blood. Casto in his report on this subject says: "The bibliographic review of this subject would indicate that hemoglobin is always reduced under anesthesia, by chloroform, ether or nitrous oxid-oxygen. Hemoglobin is markedly reduced under ether, and the greatest reduction in pigment is at the end of twenty-four hours, after

which there is a gradual return to the normal in about one hundred hours. Under nitrous oxid-oxygen anesthesia the reduction is not only slight, but the return to normal occurs in two to three hours . . . Hemolysis is observed under chloroform and ether, but no degenerated or crenated cells have been found."

Casto states further that: "The acid production of metabolism may be increased under deep anesthesia when cyanosis is permitted to occur or continue; but when a sufficient supply of oxygen is provided, this may be prevented." Yet from the experiments of others it is apparent that some cell asphyxia must result from interference with normal metabolism, regardless of the amount of oxygen in the blood.

The reports of the various writers all indicate that chloroform is the most dangerous inhalation anesthetic. While their statements may not indicate the relative clinical value of ether and nitrous oxid-oxygen, they show that the changes in the adult following the administration of ether are more permanent than those resulting from nitrous oxid-oxygen. Some observations in the present experiments suggest that the cell injury following the use of any of these anesthetics may persist for a longer period than was formerly thought possible, and leads the writer to believe that the anesthetic may be an important factor in some of the more remote postoperative complications as well as the immediate.

There is little or no experimental evidence to suggest a normal individual would die from the proper use of any of these anesthetics during the period of an ordinary operation. It seems probable that sudden deaths under ether or nitrous oxid-oxygen may result from conditions closely akin to those observed by Levy during the use of chloroform. The term, *anesthetic death,* as now used by many surgeons merely indicates that the patient died under the anesthetic, and therefore means nothing. Every surgeon should be a skilled anesthetist, and every anesthetist should have experience as a surgical assistant. Were this the case surgeons would be less prone to blame their errors in judgment on the anesthetist, and the anesthetist could better coöperate with the surgeon.

The choice of an anesthetic during pregnancy and labor involves a consideration of the fetus as well as the mother. The present experiments suggest that in considering only the dangers to the mother we may have been adding to those involving the fetus. They show that the fetus is more susceptible to chloroform poisoning than the mother, and that the long continued administration of nitrous oxid-oxygen anesthesia may asphyxiate the fetus in utero without seriously endangering the life of the mother. The danger to the fetus from nitrous oxid is apparently not completely removed by adding oxygen in the percentage found in air. Ether anesthesia seems to be better borne by the fetus in utero and the very young than is chloroform or nitrous oxid-oxygen. Therefore these experiments would suggest that ether, unless contraindicated, is the inhalation anesthetic of choice for operations during pregnancy.

Fig. 9. Liver of young guinea-pig born 23 days after 7 hours and 15 minutes of ether anesthesia and dying within 48 hours after birth. Photomicrograph x62.

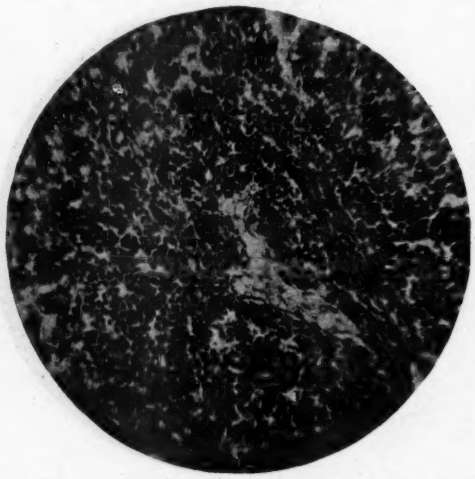


Fig. 10. The marked round cell infiltration is an indication of the primary pathology in a pregnant guinea-pig dying just before the completion of 7 hours and 45 minutes of nitrous oxid-oxygen anesthesia. Photomicrograph x288.

FACTORS DETERMINING THE CHOICE OF ANESTHETICS IN LABOR.

During labor many factors must be considered

in the choice of the anesthetic. Pain does not begin with the first evidence of the uterine contraction but at or near its height. Once pain is established little can be done to relieve the suffering from that contraction. Therefore the value of an anesthetic depends upon the quick induction of analgesia at the very beginning of a contraction. For this purpose chloroform vapor and nitrous oxid-oxygen gas are equally effective. The quick administration of ether vapor gives a considerable relief in many cases but it is less efficient than the others. However, both chloroform and ether tend to lessen the strength of the uterine contractions; they are eliminated slowly and gradually accumulate in the system; and babies born under these anesthetics may have the anesthetic in their systems for many hours after birth.

Nitrous oxid-oxygen as used in normal labor, given only during the first few inhalations at the beginning of a contraction, is very efficient and undoubtedly the safest anesthetic that has been employed. It seems probable that the fetus can absorb very little of the anesthetic during the contraction, and nitrous oxid is so quickly eliminated that very little will remain in the maternal blood at the end. Therefore, there is not the cumulative tendency of chloroform or ether, and the normal metabolism of the fetus is not disturbed during the interval between contractions.

CONCLUSIONS.

The following suggestions are offered in conclusion.

1. Chloroform, regardless of the technic employed and the amount of oxygen supplied, is the most dangerous anesthetic to both mother and fetus.

2. Nitrous oxid-oxygen anesthesia interferes with the oxygen supply of the fetus in utero and if given over a considerable period may cause its death. There is probably very little danger to the fetus during an examination or short operation if the nitrous oxid-oxygen is administered skillfully without cyanosis.

3. The continuous nitrous oxid-oxygen analgesia while less dangerous to the fetus than the anesthesia, should not be administered over long periods.

4. Ether, unless otherwise contraindicated, is the anesthetic of choice for operations during pregnancy or labor.

5. Nitrous oxid-oxygen administered intermittently at the very beginning of the contractions is the analgesic of choice for normal labor. When this is not available, ether, though less efficient, may be administered in a similar manner at a minimum of expense.

6. The term *anesthetic death* as often used means nothing.

7. There are no statistics which would indicate that the use of anesthetics to lessen the pain of labor has *per se,* increased either fetal or maternal mortality.

25 EAST WASHINGTON ST.

SOME OBSERVATIONS ON THE IMMEDIATE AND REMOTE EFFECTS OF NITROUS OXID-OXYGEN ANESTHESIA AND ANALGESIA IN OBSTETRICS.*

R. FERGUSON, M.D., F.A.C.S.,
CHICAGO, ILL.

My appearance before you this afternoon through the kind request of your Secretary, is not for the purpose of teaching you how to administer a gas oxygen anesthesia or analgesia, as I am not a specialist in this particular branch of practice. However, my thirteen years of general practice and my special work in pediatrics have lead me into the newer methods for the relief of suffering humanity, which, together with a close study of the standards of hundreds of other men in the profession have enabled me to make observations somewhat at variance with many of the currently accepted views.

My observations, therefore, are not confined to a study of my own cases, but are the product of the observations of the experiences of hundreds of men and hospitals throughout the United States and cover a period of a year and a half. During this time I have corresponded with anesthetists, obstetricians, general practitioners, both city and country, pediatrists, hospitals and nurses, while the total number of cases which this paper may be said to have covered is approximately eight thousand.

But since our limited amount of time makes it impossible to go into all phases of the subject, I shall confine my discussion to the dangers to the baby:

In order that no prejudice may enter into this discussion permit me to state that there are two classes of men interested in this important subject: (1) Those who honestly believe there is absolutely no danger to the mother or the baby neither immediate nor remote through the use of nitrous oxid-oxygen in labor, and from their experience it would seem to be so; in fact it would seem that their experience should be that of every other man who uses gas in labor. This class of men seem to be in the great majority. (2) On the other hand there

* Read during the Fifth Annual Meeting of the American Association of Anesthetists, New York City, June 2, 1917.

are those who believe there are elements of great danger not only to the mother but also to the baby, and their experience has taught them some important facts, which I wish to bring to your attention. This latter class seem to be in the minority. In order to place these opposing views before you, I shall first give you the ideas of those men whose experience has taught them there is no danger in the use of gas-oxygen in labor to the baby or mother neither immediate or remote and I shall begin with the opinion of Dr. Carl H. Davis of Chicago, who is one of the foremost users of gas in labor and has used it extensively at the Presbyterian Hospital in Chicago. To quote from his letter: "There is no apparent effect on the baby. A critical study of all cases delivered in the maternity department of the Presbyterian Hospital during a period of six months shows that the babies of mothers treated with the analgesia lose less weight during the first week than did those of mothers not given the gas. There is no variation in fetal heart tones of over five per minute and then only once in a case where gas was used for ten hours. Less effect on babies than either chloroform or ether." These observations are more or less corroborated in a degree by such men as Drs. Reed of Chicago; Barbara Hunt of Bangor, Me.; Allen of Brooklyn; Dobbin and Bergland of Baltimore; Stone of Beverly, Mass.; Allen of Colorado Springs; Meyer of Louisville; Nubbard of Boston; Sullivan of Lakeland, Fla.; Mercy Hospital, Chicago; Clark of Walter, Dakota.

Dr. Darling of Milwaukee, observed the following: "The first cry is earlier and lustier and the general condition is better than other babies." Dr. Morrison, Springfield, Ill., "No appreciable effect on baby." Dr. J. B. DeLee, Chicago, "No deleterious effect on baby for I give ether at the end of second stage." Dr. Henderson, Louisville, "No danger to baby if mother does not become cyanotic." This expression I have noted in many of my letters and please note it carefully as I hope to be able to refute the same later.

Dr. Sweet, Fresno, Cal., "No ill effect on baby before nor after delivery." Dr. Gossett, Louisville, "Absolutely no ill effect if anesthetist knows his business." Dr. R. W. Holmes, Chicago, "No effect on baby." Drs. E. I. McKesson and Dice of Toledo, "No effect on the baby." Drs. Grant, Newell, Kellogg and Irving, Boston, "Avoid cyanosis in mother and there will be no ill effect on baby." Dr. Hamilton Long, Louisville, "There is no danger to the child if the gas is in the hands of one expert in its use. Avoid cyanosis in the mother to avoid trouble in the baby." Dr. Louis Frank, Louisville, "We have had no ill effects either on mother or babe immediate or remote." Dr. W. H. Browne, Detroit, "Cannot notice any untoward effect upon the baby." Dr. Arthur B. Coon, Little Rock, Ark., "No ill effect on baby." Dr. S. Rothenberg, Cincinnati, "Even after prolonged labor, no ill effect noticed." Dr. Hoag, San Francisco, "As far as I have been able to see, there is no deleterious effect on the baby."

Dr. A. E. Guedel, "Nitrous oxid gas as applied in obstetrics has no apparent effect on either the mother or babe." (American Year Book of Anesthesia and Analgesia, 1915-16.)

From the foregoing expressions of faith in the absolute safety of nitrous oxid-oxygen in labor, and coming from so prominent and experienced men as have been quoted, it would seem that there is no room for argument. The medical literature is painfully silent regarding any untoward or dangerous effects on the baby, and until I took up the use of gas in labor, and studied its effects on the baby, and later proceeded to investigate the experiences of hundreds of men throughout this country who are using this method, I must admit that I too, believed in the absolute safety of this anesthetic, both to mother and baby, and expected it to be used unreservedly by the medical profession throughout the country. But such is not the case.

I am sure you will all understand how difficult it is to get men to admit any serious happenings in their experience. But when hundreds of men are reached by personal letters, one is certain to obtain the true experiences of some of them. This does not mean that the experiences above enumerated are not the true ones of those who were so kind as to answer my questions. But such personal letters as I have written have been instrumental in bringing out cases never reported before, and unless reported in this way would never be heard of. And if my paper will establish the fact that there are always elements of danger to the baby in the use of nitrous oxid-oxygen whether administered by an expert or a novice, and this danger is not past until twenty-four to forty-eight hours after the birth of the child; and furthermore, if it will establish a method whereby such danger may be reduced to a minimum, then my time and effort will not have been spent in vain.

Many of the men quoted above have never seen in their practice any untoward or dangerous effects on the baby, while other have expressed themselves as believing certain conditions might cause deleterious effects. There are still others who have actually seen these dangers occur either in their own or some one else's practice.

To quote further from some of my letters: Dr.

J. B. DeLee, Chicago, "No deleterious effect on the baby in my experience for I give ether at end of second stage. Do not use gas longer than three hours as there is great danger of hemolysis." Dr. E. Tuley, Louisville, "I have only seen one case in which I could attribute asphyxia of the baby to the use of gas, that one occurring recently in a multipara with second stage prolonged over an hour; gas was given fifty-five minutes; baby cyanosed, but breathed and cried promptly, and all blueness gone in ten minutes after birth." Dr. Bacon, Chicago, "When gas is given for some time before the birth of the child in a high degree of concentration, I believe that the baby is more apt to be asphyxiated than where ether is given. Occasionally I have to work sometime to resusciate the child, but have never lost one." Dr. Breitstein, San Francisco, "When 95 per cent. nitrous oxid is being given the fetal heart tones are stronger; 6 per cent. were asphyxiated but all resuscitated." Dr. Darling, Milwaukee, "Does not advocate the general use of gas unless familiar with its use." Dr. Palmer Findley, Omaha, "I regard gas analgesia as more dangerous than ether, and less satisfactory from the standpoint of expense and efficiency. More cyanosis in the babies; about 25 per cent." Dr. Brant, Boston, "We make a strong point about avoiding too deep anesthesia with cyanosis, for we believe the dangers, theoretical and clinically proven, such as increased blood pressure and decreased coagulating power of the blood, are present if at all, only when *carbonization* of the blood has taken place. As a result we are opposed to the use of oxygen and ether adding machines as offering temptation to too deep degrees of anesthesia." Dr. Hewett, Chicago, "Less asphyxia if oxygen is given at once before the cord is tied." Dr. Darling, Milwaukee, "Oxygen to mother resuscitates baby quickly." Dr. Clapp, South Bend, "While the cord is still pulsating give oxygen to mother and baby becomes pink at once." Dr. Hoag, San Francisco, "Immediately following delivery, I have always switched on pure oxygen so that most of the babies are hyper-oxygenated before the cord is tied."

Dr. A. J. Skeel, Cleveland, writing in The Jour. A. M. A., March 11, 1916, said: "I wish to enter a vigorous protest against the advice at present being so freely given that any one may use gas in labor cases with perfect safety. Gas is a powerful therapeutic agent with infinite possibilities for harm at the hands of incompetent or careless users. The statement has been made and is, I believe accepted, that gas in the hands of an expert is a safe anesthetic, but the most dangerous anesthetic if given by a novice. Even in analgesia some knowledge and experience are necessary to secure both safety and satisfaction from its use. Moreover, the temptation to follow gas analgesia labor with gas anesthesia for repair is so obvious that I would earnestly urge all those expecting to adopt this method to spend sufficient time in special study of the agents he is to use, so that he can direct its administration. Free use of gas by the inexperienced will surely lead to tragedies."

In looking back over these last quotations from so many prominent men, is it not true that we must look for some of these so-called tragedies? It is almost like a forecast of what will surely occur sooner or later. To quote from another letter, a physician, whose letter I hold in my hands: "I am glad to give you any information possible in regard to the death of my baby, and will answer your questions as follows:

(1) *What length of time was gas oxygen used?* Nitrous oxid-oxygen analgesia was given for about four hours.

(2) *Did mother show cyanosis at any time?* At no time did the mother become cyanotic; in fact, she did not lose consciousness at any time.

(3) *Was any other anesthetic used?* Small amount of chloroform was used for only a short time during delivery since gas did not seem to control the pains.

(4) *Was an autopsy held and its findings?* The autopsy was performed by Dr. Rawlson of Presbyterian Hospital, and absolutely no cause for death could be found.

(5) *Was pituitrin given at any stage of labor?* Pituitrin was given to mother but the consultant on the case could see no reason for believing the death was caused by this drug." The consultant's letter read as follows: "This baby took sick about twenty-four hours after birth with convulsions. These convulsions were recurring and the child was a picture of acidosis. Acetone odor to breath, red lips, slow pulse, respiration disturbed but not of Cheyne-Stokes type. Pulse became slower and baby died in less than forty-eight hours after birth."

This is one of the four cases I know of which has taken the same course. In all of them gas was given for several hours, more or less continuously, before delivery. No pituitrin was given in these cases to my knowledge with the exception of the first case.

Conclusions.

In studying the hundred cases in my own practice where gas-oxygen was administered by my own trained nurse, I have reached the following conclusions:

(1) That more than twenty-five per cent. of

babies delivered by gas analgesia show a slight crowing inspiration immediately after birth which may last for twenty-four to forty-eight hours. This is an entirely different condition from that encountered when mucus has been drawn into the larynx. It is more like a slight paralysis of the epiglottis with a resultant snoring sound.

(2) That this condition of a crowing inspiration is very much exaggerated where *rebreathing* is used to any great extent.

(3) That this condition of a crowing inspiration is never present for more than a few breaths if the mother is given plenty of oxygen while the cord is still pulsating, but that it may last for hours if oxygen is not given.

(4) Unusual muscular twitching lasting for about twenty-four hours but not seen if oxygen is used freely for first twelve hours after birth.

And from my study of the eight thousand collected cases here reported, with my own cases included, I have reached the following conclusions:

(1) That rebreathing is extremely dangerous to the baby and has no place in obstetrics.

(2) That air and gas are less dangerous than *rebreathing;* but slightly more dangerous than when oxygen is used and not as efficient as gax-oxygen.

(3) That nitrous oxid-oxygen anesthesia, in proper percentages for each individual case (this to be determined at the bedside), is a most wonderful help to the mother, and may even annihilate all labor pain; but that it is impossible to rule out all danger to the baby even though the mother may show no cyanosis.

(4) That nitrous oxid-oxygen anesthesia when used longer than three hours becomes dangerous to the baby by reason of the hemolysis which occurs in the mother's blood. This may occur in the baby's blood.

(5) That high blood pressure in the mother with pre-eclamptic toxemia is a contraindication to the use of gas-oxygen. Under these conditions it is not only dangerous to the mother, but also to the baby.

(6) That it is never safe to use gas where the mother is suffering from hyperthyroidism.

(8) That every baby showing a crowing inspiration at birth, or where the mother during labor has complained of a marked dryness of the throat while taking nitrous oxid, should be allowed to absorb all oxygen possible from the mother before the cord is tied, the mother receiving pure oxygen until cord stops pulsating.

(9) That the babies are safe for three hours of a gas labor providing mother is never allowed to become cyanosed and nitrous oxid is not used above eighty per cent. the other twenty per cent. being oxygen, not air.

(10) That when gas is used longer than three hours, there is great danger of a condition developing in the baby simulating the late deaths from chloroform or ether narcosis: and perhaps identical with the conditions we call acidosis.

(11) That when gas is used in a high degree of concentration for longer than three hours, the baby should receive not only all the oxygen possible before the cord is tied, but it should be further resuscitated by the giving of pure oxygen, more or less continuously, for from twenty-four to forty-eight-hours after birth. This should be done as a routine in order to avoid a condition simulating the cases of postoperative narcosis from ether and chloroform and which, for want of a better term I have called *acidosis.*

(12) That the expression *carbonization of the blood,* used by so many men to describe a theoretical condition in the baby, is a misnomer and means absolutely nothing. My authority for this statement is Prof. Carlson of the University of Chicago. The term should be supplanted by an expression which would better describe the true state of the blood. And since carbon dioxid and nitrous oxid do not in any way combine with hemoglobin in the blood, but only starve the body cells of oxygen and thereby cause retention of the acid products of metabolism, might not this condition in the baby or even in the mother be properly called *nitrous oxid acidosis.*

(13) That my correspondence shows conclusively that the following quotation from Palmer Findley, of Omaha, is almost, if not, absolutely correct: *"I regard gas analgesia as more dangerous than ether, and less satisfactory from the standpoint of expense and efficiency; and that about twenty-five per cent. of the babies are cyanotic."*

(14) That many general practitioners, especially those in the country far away from hospitals and assistants have already discarded their gas machines as being too cumbersome and expensive, as well as inefficient and dangerous when compared to the almost perfect safety of ether.

(15) That the following expression of Guedel from the Year Book of 1915-16, that "the time will soon come when this gas may be left with the nurse or experienced attendant, and the woman in labor be enabled, by self administration, to secure relief safely with analgesia for any number of hours, leaving the physician free to come and go at will," is far from realization and probably will never be realized.

ABSTRACT OF DISCUSSION

DR. JOHN OSBORN POLAK, Brooklyn, N. Y.: I have been particularly interested in Dr. Davis' researches and in

Dr. Ferguson's record of immediate and remote effects of nitrous oxid-oxygen on the child. Too much emphasis cannot be placed on the dangers of delayed chloroform poisoning, the incidents of which has again been so strikingly demonstrated in Dr. Davis' guinea-pig experiments. In instances of chloroform poisoning large amounts of the drug have usually been given and its occurrence may not occur for some days. We have been using gas-oxygen in our clinics, as a routine analgesic in labor for a period of two and a half years. We can indorse and confirm much of what Dr. Ferguson has developed in his canvass. The difficulty of hitching the anesthetic to the obstetrician may account for the success of one method in a certain clinic and its failure in another. When scopolamin-morphin narcosis for obstetrical amnesia was introduced in this country there were a number of deaths on account of prolongation of the second stage of labor. In these instances it is not the anesthetic that produces death, but the changes in the child due to the prolonged labor. Of course in any prolonged labor the anesthetic also causes complications, especially chloroform. Labor should be so conducted that the second stage is not prolonged, for if it several hours, even under gas-oxygen, you will get a cyanotic baby, with a great deal of mucus in its airway, and it will have to be resuscitated. A prolonged second stage under ether-oxygen, or without any anesthetic would similarly effect the baby. The three methods of anesthesia, now in routine use, each have their place in the conduct of painless labor. Scopolamin-morphin is ideal for the first stage, gas-oxygen for the second stage and for complete labor in uncomplicated cases; and ether-oxygen is the anesthetic of choice for operative obstetrics. While none of these methods are absolutely safe, in the sense that they cannot be used with impunity, they do not injure the mother, and if used judiciously, the baby can be delivered in as good condition as if no anesthetic had been used.

Dr. Charles K. Teter, Cleveland, Ohio: Nitrous oxid-oxygen anesthesia is not an obstetrical panacea. However, my continued use of the method, since presenting the subject in my Presidential address before this Association two years ago in San Francisco, convinces me that the method is a good one in its proper place. In consulting Dr. A. J. Skeel, who has used the method extensively, he informed me, just before this meeting, that he was obviating the occurrence of perineal lacerations by the addition of ether. The use of nitrous oxid alone or with air may stimulate the respiratory efforts of the baby and account for the excessive mucus in the baby's throat. All cases of cyanosis have readily responded to oxygenation. Dr. Davis is to be congratulated on his researches in the physio-pathology of these various methods of anesthesia and adding so much to the sum total of our knowledge on the subject.

Dr. John R. Worley, Dallas, Texas: I have been using gas-oxygen analgesia and anesthesia as a routine in labor for some years, and in a paper, recently read before the Gynecological Section of the Texas State Medical Society, I went so far as to state that there were no contraindications to its use, and that in my personal experience I had seen no detrimental effects on either the mother or baby, and have seen more pulmotor resuscitation on the other fellow's babies, in labors in which no anesthetic had been employed. I have seen no dead babies in ordinary deliveries under gas-oxygen. We have lost babies in deliveries requiring forceps or surgical interference, especially in breech presentations. In one patient, with a cardiac complication, nitrous oxid-oxygen was used for eight hours during labor and this patient and baby survived and are now living. We lost the baby in a breech case that lasted 18 hours. In reference to the effects of nitrous oxid-oxygen on the fetus in utero, I have never known of a pregnant woman, coming to operation under this anesthetic, for some surgical procedure, to abort. Not long ago one of my confrere's wife, five months pregnant, was operated under gas-oxygen and a cystic ovary was removed. She went to term and was delivered of a healthy child, now living.

Dr. R. Ferguson (Closing): In investigating the clinical side of this subject it was my object to find out if gas-oxygen was safe in every individual case. The records I have collected speak for themselves, and from these observations, as well as Dr. Davis' researches, it must be concluded that there are contraindications to the use of nitrous oxid-oxygen anesthesia and analgesia in labor and dangers, especially to the baby, which must be guarded against, before it can be called entirely safe.

Dr. C. Henry Davis (Closing): Clinically, in its use in many cases, I have found nitrous oxid-oxygen safe and harmless in labor; but I fully realize its theoretical dangers, and the fact that other methods of anesthesia have their place in obstetrics.

VASCULAR REFLEXES WITH VARIOUS TENSIONS OF ETHER VAPOR.*

Frank C. Mann, M. D.
Rochester, Minnesota.

The study of ether percentages by Boothby,[1] who showed that in man all individuals become anesthetized at a definite tension of ether, and the introduction by Connell,[2] of the anesthetometer, an instrument adopted to accurately administer definite and known tensions of ether, have opened a new field for the investigation of physiological phenomena in relation to anesthesia. This is of importance, not merely in regard to the process of anesthesia but also in regard to the many physiological problems, the study of which compels the use of an anesthetic.

The physiological investigator employing anesthetized animals has usually been content to state that the animal was lightly anesthetized or deeply anesthetized as the case might be. While this may mean a fair degree of accuracy by each individual investigator for any particular series of experiments it certainly does not mean the same in all instances because what might be a lightly anesthetized animal to one would appear deeply anesthetized to another. Furthermore, difficulty is experienced in attempting to corroborate the work of any investigator because of the anesthetic factor. This has been repeatedly emphasized by Boothby.

The non-standardized state of anesthesia in regard to physiological research has probably been partially responsible for contradictory data concerning different problems. It has undoubtedly been a factor in the shock problem and may be responsible for some of the debated problems in regard to blood pressure and respiration.

The present research was undertaken to determine to what degree blood pressure and the vascular reflexes are modified by the different tensions of ether, and to establish an optimum range of tensions for these reflexes.*

* Read during the Fifth Annual Meeting of the American Association of Anesthetists, New York City, June 2, 1917. From the Department of Physiology, Mayo Clinic, Rochester, Minnesota.

*When I first thought of working with the Connell apparatus I intended to establish a standard of ranges of anesthetic tension for each species of the common laboratory animals. One range would include the optimum tension for physiological work and another the tensions which are liable to prove fatal. In a discussion of the subject with Dr. W. Boothby he suggested that I finish a

The particular Connell apparatus obtained was made by a new manufacturing company and was not mechanically correct. When calibrated by the method described by Boothby and Sandiford[3] it was found to deliver a tension considerably less than it was supposed to be standardized for and there was a wider range of variation than desired. It is hoped that an instrument which can be calibrated as closely as Dr. Boothby's will be secured for finishing the problem.

The tensions found necessary for work on the dog as used on the apparatus were between 40 and 64. These available tensions when calibrated by means of the Waller gas balance were found to have approximately the following values:

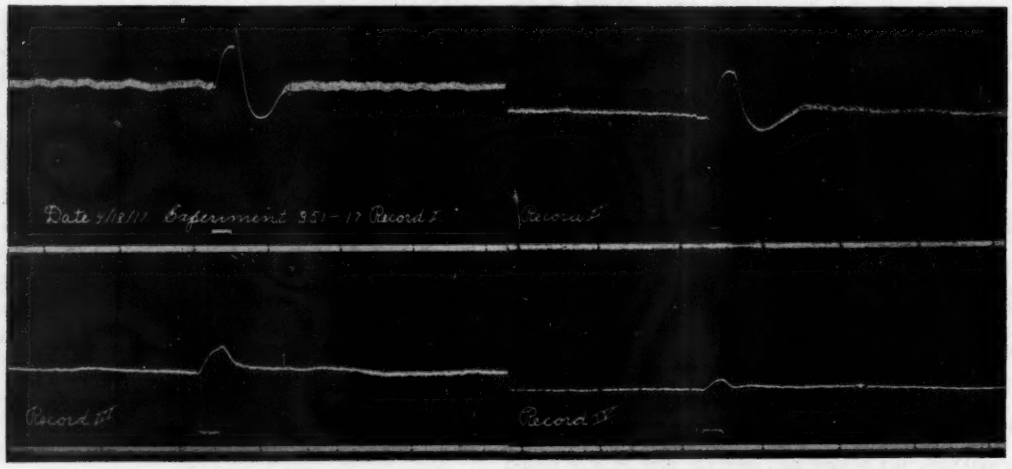
Tension on Connell.	Tension on Waller.
40	36.4
44	38.1
47	43.6
51	44.1
53	49.2
54	48.4
58	50.7
61	54.5
64	55.1

I am indebted to Miss Sandiford for invaluable aid in calibrating the apparatus.

problem which he had started with Dr. W. T. Porter, in regard to the vascular reflexes with various tensions of ether. Unfortunately Dr. Boothby was called to the war before the work was completed. For this reason, as well as the fact that the particular instrument used was found to have a rather large coefficient of error, this paper must be considered as a preliminary report.

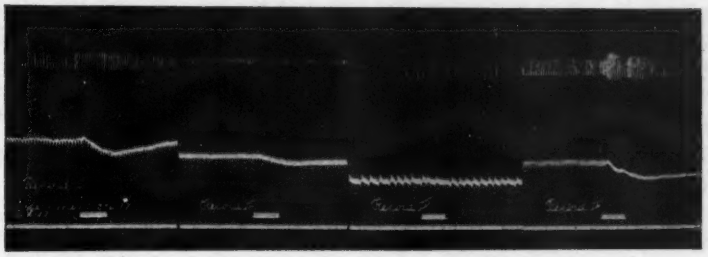
In the calibration no account was taken of the alcoholic content of the ether and the apparatus was run in practically the same manner as used in the experiments. While there was considerable variation the actual tension of ether must have been very nearly that computed on the Waller balance. Owing to this wide variation of tensions due to the apparatus, only approximate tensions will be given now and later I hope to check them with a more accurate instrument. The experimental results will be expressed in approximately the corrected tensions.

All experiments were performed on dogs. The animals were etherized by means of a cone or cabinet and after intubating a definite tension of ether was administered. This was usually the lowest tension compatible with operative work. The apparatus was arranged to record carotid blood pressure (mecury manometer) and respiration. The desired nerves were carefully prepared in the usual manner and all precautions were taken to preserve them in a normal condition. All stimulating was done with a platinum or shield electrode and a Kronecker inductorium using a primary current derived from storage batteries. While no effort was made to standardize the stimulus, great care was taken to keep it constant for each experiment. When pressor reflexes were desired, the strength of stimulus and rate were made far above the threshold for these reflexes. Both were decreased for depressor reflexes.



Experiment 351-17. Kymograph record showing effect of various tensions of ether on the pressor reflex of the vagus. There was an interval of one hour between each record. Nerve stimulated: Central end left vagus; right vagus sectioned.

	Ether tensions (Approximate)	*Blood pressure reaction.*
Record I	36	Increased from 138 to 194 then dropped to 105.
Record II	43	Increased from 104 to 150 then dropped to 90.
Record III	48	Increased from 54 to 80, maintained.
Record IV	50	Increased from 35 to 48, maintained.



Experiment 395-17. Kymograph record showing effect of various tensions of ether on depressor reflex of sciatic. There was an interval of one hour between each record. Nerve stimulated: Central end right sciatic.

	Ether tensions (Approximate)	*Blood pressure reaction*
Record I	36	Decreased from 100 to 78.
Record II	43	Decreased from 80 to 70.
Record III	44	No change.
Record IV	Air	Decreased from 70 to 50.

Note that the reflex disappears under a relatively low tension.

Vascular reflexes obtained in an animal with intact respiratory mechanism are affected by the changes in respiratory movements. To obviate this curare was used in a few experiments but it was found that even with the greatest care the product which can be obtained now was too toxic. However as this is only a comparative study for each animal the respiratory factor is not great enough to seriously affect the general results.

One hour was arbitrarily chosen as the time necessary for the ether in the tissues to reach the same tension as in the alveolar air. From the careful observations in these experiments I believe that one hour is fully ample. Care was taken not to fill the apparatus within 15 minutes before stimulating.

After administering the anesthetic at a constant tension for one hour the nerves were stimulated for a definite time, usually fifteen or thirty seconds, and the changes in blood pressure and respiration recorded. Then the ether was changed to another tension. After all the desired tensions were used the animal was either given air or a very low tension and the nerves stimulated. This was done in order to be sure that the nerves were still functionating. However, the results show that on the whole this was not necessary because in only two experiments were the nerves found to be badly injured.

In general, the physiologic phenomena occurring under ether were found to be remarkably constant at the same tension in different dogs.[4] While the effects of different tensions upon blood pressures, the vascular reflexes, respiration and the respiratory reflexes were not always progressive or absolutely constant, yet there were certain narrow limits of tension within which certain changes always occurred. However, with a very accurate apparatus I believe all the reflexes will be found to be proportioned to the anesthetic tension used.

It was found that the lowest tension compatible with operative work on the dog was approximately 36. At this tension all the operative procedures necessary for the experiment could be carried out but the animal was always very lightly anesthetized and it was usually necessary to administer the ether for over half an hour at this tension before beginning work. This tension was too low for satisfactory routine work.

The optimum tensions for operative work are probably between 38 and 45. At these tensions the animal reacts slightly to operative procedures but the condition remains excellent. As the ether tension is increased above 36 the animal progressively exhibits the signs of deepening anesthesia but the condition remains good until a tension of 48 is reached. The range of ether tension between 36 and 48 is the range of surgical anesthesia in the dog. While blood pressure and respiration may both be greatly depressed it seems impossible to kill an animal with any tension within this limit provided time is not allowed to become a factor.

When the tension is increased above 48 the animal becomes profoundly anesthetized and respiration is liable to fail and blood pressure to decrease to a very low level. This may occur at any tension above 48 and 60, but usually it takes place at tensions between 50 and 55. In all probability a dog cannot withstand a tension above 60. None in my series has ever been able to withstand a tension of 60. The range of tensions between 48 and 60 is the limit of viability for the dog. Any tension within this limit may prove fatal to the animal.

[4] This corresponds to the results of Boothby in man.

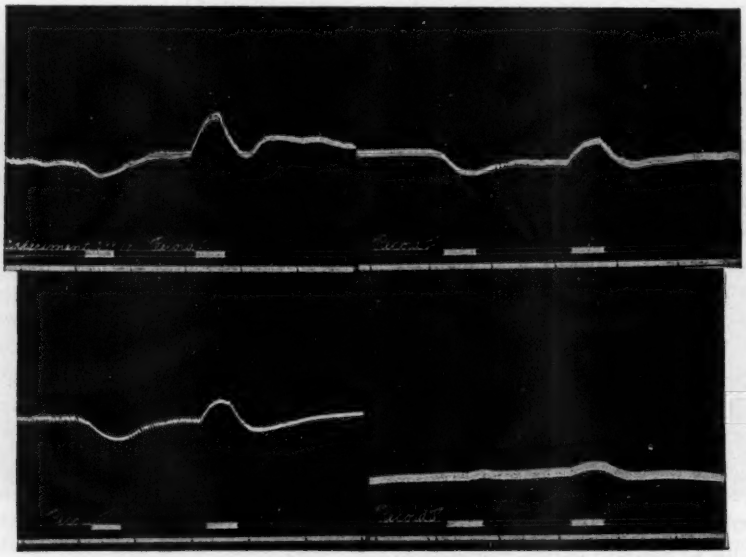
The corneal reflex is very active at tensions 36 to 40. Under a tension slightly above 40 it may disappear. It probably never persists at tensions above 45.

The blood pressure usually varies considerably under a tension of 36 because of the lightly anesthetized state of the animal. Under all increased tensions it remains very regular for that tension. It may remain at about the same level or be successively slightly decreased at each increase of tension until a tension of 43 is reached. It is always greatly decreased by tensions above 48 but usually does not reach zero at tensions less than 50 and rarely persists at a tension of above 55.

In a few experiments the blood pressure slightly increased as the ether tension was increased until 48 to 50 was reached when it decreased very markedly.

The changes in blood pressure under the different tensions of ether depend somewhat upon the initial pressure. When the pressure under 36 is high each increase in tension may produce a definite decrease in the pressure. In the experiments in which the pressure under 36 was low, the first few increases in the ether tension did not always decrease the blood pressure. However, there was always a marked decrease in the pressure when tensions of 48 or 50 were reached.

Not only is the blood pressure not depressed to any great extent by the lower tensions, 36 to 45, but it quickly recovers even after the animal has been etherized for many hours. This is not true for the higher tensions. After an animal has been under a tension between 48 and 55 for an hour or two, blood pressure is greatly depressed and even though ether be withdrawn and air administered until the anesthesia becomes so light that it is necessary to administer more ether, blood pressure only partially



Experiment 399-17. Kymograph record showing the effect of various tensions of ether on depressor and pressor reflexes produced by stimulating the same nerve with different strength and rate of stimuli. There was an interval of one hour between each record. Nerve stimulated: Central end of left vagus. Right vagus sectioned. Stimulus: Primary current derived from storage battery. Depressor response produced with secondary coil at 16, rate 4. Pressor response produced with secondary coil over primary, rate 24 to 30.

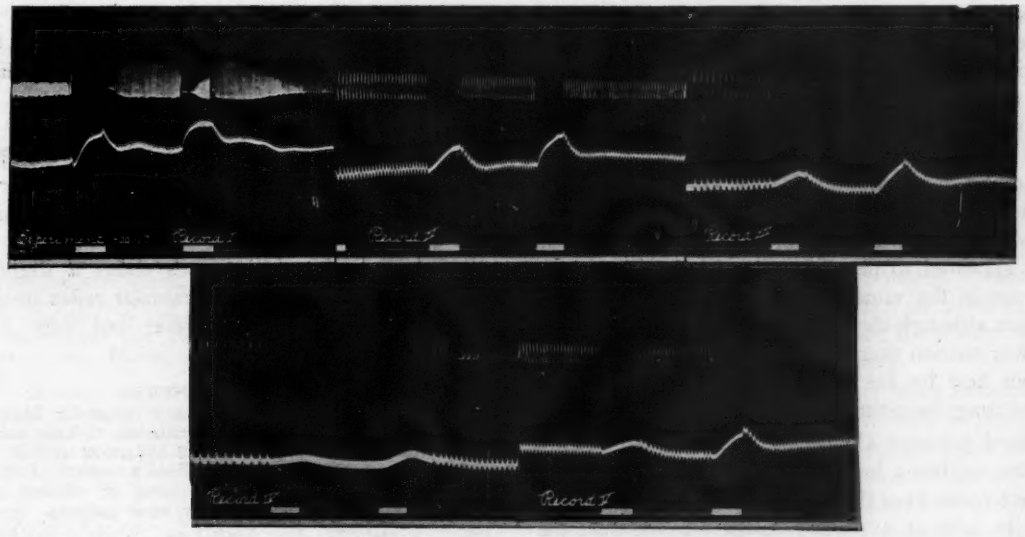
	Ether tensions (Approximate)	*Blood pressure reaction*
Record I	36	Decreased from 108 to 92. Increased from 120 to 166.
Record II	43	Decreased from 120 to 94. Increased from 105 to 130.
Record III	44	Decreased from 130 to 108. Increased from 128 to 150.
Record IV	48	Barely perceptible rise. Respiration gone. Increased from 60 to 66.

Note that the depressor reflex and respiration disappeared while the pressor was still active.

recovers. This result was probably due not only to the anesthetic but also to the long continued low blood pressure.

For the purpose of this study the vascular reflexes may be divided into four groups. The first division is that of pressor and depressor. Stimulation of any mixed nerves will produce these reflexes. When the stimulus is relatively strong and the rate rapid, pressor responses are elicited. When the stimulus is weak and the rate slow depressor reflexes are produced. Each of these reflexes may be divided into two groups, depending upon their path. tion of the central end of the vagus is the easiest of the vascular reflexes to obtain in the dog and for that reason it was studied the most extensively. This reflex is greatest at tensions between 36 and 45. In most instances it is decreased under tensions above 45 although a well-marked rise usually occurs under all tensions up to 48. It is either gone or barely perceptible at a tension of 50 and rarely persists at all above this tension.

Like the blood pressure, it is quite common for the reflex to progressively decrease under each increase of tension until it finally disappears. But this is



Experiment 400-17. Kymograph record showing effect of various tensions of ether, on the vascular and respiratory reflexes of the vagi. There was an interval of one hour between each record. Nerves stimulated: Central ends of right and left vagi.

	Ether tensions (approximate)	*Blood pressure reactions*
Record I	36	Increased from 104 to 140 and 122 to 158.
Record II	43	Increased from 94 to 120 and 100 to 132.
Record III	44	Increased from 70 to 86 and 70 to 94.
Record IV	48	Increased from 55 to 60 and 54 to 65.
Record V	36	Increased from 72 to 80 and 70 to 92.

Note that besides a depression of the pressor vascular reflex by the increased tensions of ether there is an increased response to the reflex inhibition of respiration.

In a mixed cerebral nerve the number of neurones between the one stimulated and the vasomotor center must be few. In all the other nerves at least for pressor reflexes there are a large number of intermediary neurones. The vagus was taken as the example of a nerve with few intermediary neurones while the sciatic was taken as representative of those having many intermediary neurones. In each instance the nerve was sectioned and the central end stimulated, care being taken to stimulate in the same manner each time.

The pressor vascular reflex produced by stimula- not constant and in several experiments the reflex might be slightly greater at the higher tension. However, the range of tensions under which it is well marked and the limits at which it disappears are very constant.

The character of the reflex also changes with the change of ether tension. Under the lower tension the rise is not only greater but it is sustained for a greater length of time or a depressor effect takes place immediately after stimulating. As the animal becomes more deeply etherized under the higher tensions the blood pressure does not increase

so much but quickly returns to its former level upon cessation of the stimulus. After an animal has been under the higher tensions for some time and the anesthetic is then withdrawn until the anesthesia becomes very light, the vascular reflex is usually well marked but not so great as it was initially under a tension of 36.

The pressor vascular reflex produced by stimulation of the central end of the sciatic is depressed by all tensions above 45 and does not appear to persist at tensions above 50. In this respect it is very similar to that of the vagus except that it is more greatly depressed by increased tensions and probably disappears sooner.

Depressor reflexes of either the vagus or the sciatic are quite difficult to obtain in the dog. For this reason the deduction of definite conclusions in regard to the depressor reflex should not be made until comparative data are obtained also in the cat and rabbit in which animals these reflexes are easily elicited. However, in the experiments on the dog in which I was able to obtain depressor reflexes they appeared to parallel quite closely the pressor reflexes in the same nerves under the same ether tension although they sometimes disappeared under a lower tension than the pressor reflex. This was shown best by the depressor reflex produced by stimulating the central end of the vagus.

Blood pressure always persists at either a high tension or for a longer period of time under the same tension than the respiration. The respiration usually fails at a slightly lower tension than the vascular reflexes although in some cases this reflex fails first.

CONCLUSIONS.

These conclusions dealing with the vascular changes under different tensions are based upon experiments performed upon the dog, the Connell apparatus being employed.

1. In general, the physiologic phenomena due to ether are remarkably constant at the same tension in different dogs.

2. The lowest ether tension which will allow operative work is 36.

3. Any tension between 36 and 45 is safe as regards the life of the animal.

4. Any tension between 48 and 60 is liable to prove fatal.

5. Probably no individual dog can survive a tension of 60.

6. The corneal reflex does not persist at tensions above 45.

7. Blood pressure may remain practically the same or be successively slightly decreased at each increase of tension up to 48. It is always greatly decreased by tensions at or above 50. It reaches zero under tensions between 50 and 60.

8. The pressor vascular reflex produced by stimulation of the central end of the vagus is greatest at tensions between 36 and 45. It is decreased under tensions above 45 although a well-marked rise usually occurs under all tensions up to 48. It is either absent or only barely perceptible at a tension of 50 and rarely present at all above this tension.

9. The pressor vascular reflex produced by stimulation of the central end of the sciatic is depressed by all tensions above 45 and does not persist at tensions above 50.

10. The pressor reflex of the sciatic seems to be depressed more greatly by increased tension than that of the vagus.

11. In the instances in which it was possible to study the depressor reflex it appeared to parallel quite closely the pressor reflex in the same nerve under the same tension although it did not persist under quite as high tensions.

12. Respiration usually ceases under a slightly lower tension than the pressor vascular reflex of the vagus disappears although either one may fail slightly before the other.

ABSTRACT OF DISCUSSION.

DR. F. W. NAGLE, Montreal: I have found Dr. Mann's paper exceedingly interesting and valuable. I have noted his same difficulty with the Connell apparatus and in its use during the past year I have utilized pressures of from 50 to 70, the lower pressure providing an efficient anesthesia only for children and very weak patients.

DR. S. J. MELTZER, New York City: While it has been stated that the same tension of ether in all individuals produces the same effects, it must be understood that no matter how accurately an apparatus has been manufactured and adjusted, that animals and individuals vary in their susceptibility to anesthetics, and consequently the biological element in percentage methods of anesthesia remains variable and occasionally disconcerting. The apparatus is reliable enough but the individual remains unstable. Thus tensions and pressures can only be approximated in relation to their usual results.

DR. VAN HOUSEN, Chicago: Dr. Mann's investigations offer a scientific explanation of why ether and chloroform, following scopolamin-morphin narcosis, should never be pushed to the point of abolishing reflexes. I attribute the success of my instruction in the use of this method of combined anesthesia and my experience with it to emphasizing the fundamental principle that in scopolamin-morphin narcosis the reflexes are never abolished and that in supplementing such narcosis for greater relaxation, with chloroform or ether, the reflexes must not be abolished. If the reflexes are obtunded the supplemental anesthetic must be withdrawn until they again become active.

DR. F. C. MANN (Closing): Working with a somewhat inaccurate apparatus makes my conclusions open to question, but I feel justified in holding that within limited range of tensions you get the same phenomena in all individuals. In animal experimentation one can readily sense the variations in response to different tensions, and I am confident it can be established, with a more accurate adjustment of the apparatus, that within a limited range of tension, all animals of a species will respond similarly.

The response in different animals is marked. Thus goats, which are exceedingly difficult to anesthetize on

account of hypersecretion of mucous and are prone to anesthetic fatality during experimentation, will run under very much less tension than dogs.

I found also that ether, as near as I can determine, blocks all nerve reflexes except one, provided you give a high enough tension. As the tension of ether is increased the vascular reflexes decrease and a tension is reached under which they do not occur. The pressor reflexes of respiration, those which you wish to excite when you intend to bring the patient back who has been over-anestetized, will also disappear in a majority of cases slightly before respiration ceases. However, there is one reflex which I have never seen blocked in any anesthesia. Any reflex which will inhibit respiratory movements under light anesthesia will continue to do so under any tension until respiration ceases. This will occur even if respiratory movements are only taking place three times a minute. That is in connection with the point that Dr. Muns made this morning in regard to depressor reflexes. Certainly the depressor reflexes disappear slightly quicker than the pressor ones did, and that is more particularly true in regard to respiration. On the other hand, as the anesthetic tension becomes greater, and the animal becomes more under the influence of the anesthetic, the pressor reflex is not obtained. You can stimulate any or all nerves, but it is not obtained and I doubt very much whether respiratory stimulation will produce any beneficial effects on blood-pressure.

REFERENCES.

1. Boothby, W. M.: Ether Percentages. Jour. Am. Med. Assn., 1913, LXI, 830-834.
2. Connell, K.: An apparatus (Anaesthetometer) for Measuring and Mixing Anesthetics and other Vapors and Gases. Surg., Gynec. and Obst., 1913, XVII, 245-255.
3. Boothby, W. M., and Sandiford, I.: The calibration of the Waller Gas Balance and the Connell Anesthetometer. Jour. Pharm. and Exper. Therap., 1914, V, 369-378.
4. Boothby, W. M.: The Determination of the Anesthetic Tension of Ether Vapor in Man, with some Theoretical Deductions therefrom, as to the Mode of Action of the Common Volatile Anesthetics. Jour. Pharm. and Exper. Therap., 1914, VI, 379-392.

THE EFFECTS OF ETHER ANESTHESIA AND OF VISCERAL TRAUMA AS SHOWN BY VASOMOTOR AND BLOOD-PRESSURE CHANGES. *

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These experiments, which have been reported elsewhere[1] were undertaken with a view of determining the actual circulatory changes taking place in the organism during long continued third stage ether anesthesia without other procedures, and also during maintained visceral irritation in the abdomen, the subject being under ether anesthesia.

There are many vexing and moot questions that come up in the consideration of circulatory changes during ether anesthesia. Investigators agree generally that in ether anesthesia there is first a rise in blood pressure corresponding to the first or excitement stage of anesthesia which is followed by a distinct lowering of pressure in long continued third stage anesthesia. Indeed it is a fundamental property of anesthetic substances that they produce the phenomena of excitation before that of depression. Concerning the causes of lowered blood pressure in third stage anesthesia writers do not agree. There are those who maintain that local action of ether on the heart is responsible for the hypostasis, while others see in the phenomena a central as well as a local vasomotor effect.

The condition of the peripheral arteries during third stage ether anesthesia also provokes disagreement. While most all writers agree that the smaller arteries and capillaries are dilated, the opinions diverge on the cause of the action. Some claim a central medullary depression with peripheral paralysis, while others see a direct local dilatation.

Visceral irritation such as experimental intestinal trauma or ordinary surgical operations under ether anesthesia is accompanied by certain circulatory changes. All workers, of course, agree on the pure observable phenomena. While a low blood pressure in shock is a common observation, the theories as to its cause are quite divergent and have caused a great deal of discussion. Against those who proclaim exhaustion and paralysis of the vasomotor center as the cause are those who maintain that other factors are at work.

Inseparable from the subject of blood pressure in shock, is the question of the condition of the peripheral arteries. The greatest disagreement exists between observers as to whether the smaller arteries and arterioles are dilated or contracted in shock. Whether they are contracted or dilated there seems at present to be no chance of agreement as to the reasons for either phenomenon.

While it is impossible to answer all of these questions by deductions from a relatively few graphic experiments, it may be possible to at least set at rest a few doubts as to the purely observable phenomena in connection with the circulatory system.

It is proposed therefore to graphically demonstrate the actual changes in the blood pressure, and in the vasomotor reactions during ether anesthesia, and those variations accompanying visceral trauma under ether. Such experiments may show the definite relations existing between the blood pressure and the vasomotor status, and also the relationship between the depression and the mechanical stimulation of the visceral manipulations. The question naturally arises as to what depressing, exciting, or neutralizing effect the anesthetic may have on the vasomotor mechanism in surgical manipulation of viscera.

Dogs weighing 6 kg. to 12 kg. were used. The ether was administered by intratracheal tube through an ordinary breathing bottle in half the cases, and

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by continuous insufflation in the other half. The blood pressure was taken from the carotid and recorded through a mercury manometer. One hind leg of the dog was placed in a plethysmograph of large size; the apparatus was then connected with a recording water manometer and the whole system filled with water. Every change in volume of the leg denoted a variation of the peripheral vasomotor mechanism within the leg.

In one series of experiments many dogs were caused to remain under ether for long periods of time, records being graphed every half hour. The shortest experiment was one hour in length and several experiments lasted six hours and over. In several cases there was a slight fall of blood pressure after the first half hour. In one-half of these cases the fall in pressure continued to death, and in the other half there was a slight gradual rise to death. In those cases showing rapid fall of blood pressure, the animals died in a short time. The dogs that showed a gradual rise of pressure remained alive six hours and over. In five to forty minutes after the beginning of the experiment, a change in leg volume was noted. Several cases showed a vasodilatation and several showed a vasoconstriction. At the end of the first half hour there was a general vasodilatation shown in all cases but one. At the end of the first hour (the second hour of anesthesia), all cases showed a marked vasodilatation, which condition slowly increased, with occasional periods of vasomotor recovery, until at death there was a total increase of leg volume of 2 cc. to 18 cc.

The normal tone and resistance in the arteries were altogether or partly destroyed in all cases, and the blood pressure lowering effect of this one factor, obtaining throughout all the periphery, must have been tremendous. Why, then, was there in half the cases a decided and fatal fall in blood pressure, and in the other half a decided maintenance of the tension? It is evident that the question must be answered by considering the normal compensatory reaction of the heart to the blood pressure lowering tendency of the peripheral dilitation. While the heart did not increase in rate in any instance, there was a decided increase of ventricular output, an increase great enough to overcome the hypostatic effect of the peripheral dilation: the blood pressure was maintained and even raised a little. It is evident that if the heart fails to show this compensatory reaction, because of organic disease, or too early response of the nervous centre to the effect of the anesthetic, the blood pressure must fall, as it did in the three cases noted.

In considering the cases showing the failure of the circulation, it is permissible to argue that any means of preventing or overcoming the peripheral vasodilatation would have helped to maintain the blood pressure.

In summing up the results of these anesthetics we may say that (1) ordinary third stage ether anesthesia prolonged beyond one hour results in marked vasodilatation of peripheral vessels; (2) that there is a direct relationship between the condition of the vasomotor control and the blood pressure; and (3) that the end result of ether depression is loss of vasomotor function and death.

In another series of experiments the dogs were anesthetised after which the whole small intestine was delivered through an incision in the linea alba. The intestine was then kneaded and rolled between the hands or in a towel constantly for varying lengths of time, deep anesthesia being maintained. The experiments lasted from 45 minutes to 165 minutes after adjustment of the apparatus. The deep anesthesia and the violent trauma brought on death quickly. In all cases but two the blood pressure was maintained throughout the experiment, or at the end was higher than at the beginning. It was only at the very last minute of life that the loss of vasomotor function occurred and the blood pressure fell rapidly to death. In all cases but one, there was a definite vasoconstriction of peripheral arteries which resulted in a fall of leg volume of 2 cc. to 10 cc. This change in the peripheral vessels began almost immediately the intestines were disturbed and continued as long as the stimulation was applied.

The vasoconstriction in the peripheral vessels seemed to be an important factor in maintaining the blood pressure in cases of gradually developing collapse from ether depression, as it may be in cases of lowered blood pressure from splanchnic dilatation in traumatic shock. Whenever the intestinal irritation was not accompanied by vasoconstriction of the peripheral vessels, the blood pressure tended towards a fall, through the vasomotor depressent effect of the ether. Whenever the vasoconstriction was present *but slightly*, the blood pressure showed itself to be better maintained, the depressor and pressor agents offsetting each other. When the vasoconstriction was marked, the tendency towards the maintainance of the general blood pressure was greater. In some cases there was an actual rise of blood pressure due to the domination of the pressor over the depressor effect.

In summing up the results of these intestinal trauma experiments we may say that (1) manipula-

tion of the intestines brings about a vasoconstriction; (2) that there is a relationship between this vasoconstriction and the blood pressure.

In the light of what we have learned from these and other experiments, it may be said that an anesthetic substance such as ether and an irritation to abdominal viscera such as intestinal handling, produce two different kinds of stimuli. The anesthetic substance first excites and then depresses. The depression is its essential and marked effect and if too prolonged it results in total functional incapacity. The effect of visceral irritation is wholly excitatory, being mechanical. When this irritation becomes too severe or is continued too long the summated excitant stimuli leads to over-activity and fatigue, and the end of fatigue is exhaustion.[2] Then both the anesthetic and the visceral injuries may lead to absolute conditions, either complete depression or complete exhaustion. But, though the end result is reached by entirely different processes, the effect is identical in a complete functional incapacity. An exhausted function is just as incapacitated as a depressed function.

In the ordinary major operation with ether as the anesthetic, a moderate amount of excitation from handling is a helpful factor. There can be no question that there are occasions, when the patient is on the verge of syncope from ether depression, when a vigorous cutaneous or visceral irritation would restore the vasamotor tonus, resulting in a beneficial reflex rise in pressure. There are many experiments that show the sensitiveness of the vasomotor mechanism to reflex stimulation, and it is clear how such a reflex stimulation in proper time and moderation may very well become a remedial measure in impending ether death. There are a few reported cases where the sudden cessation of manipulation of the peritoneum during laparotomies resulted in arrest of heart and respiration. In such cases a certain amount of irritation was balancing a certain amount of anesthesia, and when the irritation was suddenly removed, the anesthesia slid into the danger phase. When sensory stimuli and the anesthetic are exhibited together there is one period when the excitation from the surgical technic and the depression from the drug are pitted against each other to the good of the patient. Either may be better borne in the presence of the other.

If the anesthetic and its neutralizing irritation be continued too long or are of undue severity, the two antagonists become allies in effect and bring about a much quicker collapse from functional incapacity than had only one agent been at work. It is suggested therefore, that the symtom complex known as post operative shock is a combination of the effects of excitation and depression and varies directly with these two factors.

If these deductions form the true explanation of the relationship and sequence of events in ordinary surgical operation that go on to post operative shock, the present day ideas of prevention and treatment of such conditions must be altered. If one can accept the fact that ether anesthesia and visceral trauma may each in itself cause functional incapacity, and that when applied together they may act quicker and surer than when used alone, there can be no crimination as of surgeon against anesthetist, or as of anesthetist against surgeon in case of accidents. If the prevention of post operative shock depends upon a proper balance between the two types of stimuli, then both surgeon and anesthetist should quite properly understand such relationship, and so govern the administration of the two types of stimuli that they will not be too severe, too long continued, or too out of balance. The importance of this idea is being more and more recognized.

If functional incapacity may be brought about through the excessive application of either type of stimulus, or still easier by the use of both types together, the treatment of post operative shock must be based on the proper designation of the predominating etiological factor. It is obvious that remedial measures designed to counteract a condition brought on by too much ether might not be exactly suited to cure traumatic or sensory shock. Although both conditions are expressed outwardly in the one common way the body mechanism can display them, the absolute condition is reached by entirely different processes.

BIBLIOGRAPHY.

[1] Proc. Soc. Exper. Biol. & Med., 1915, xii, 87-90. Journal Missouri State Medical Associations, 1915, xii, 482; and Annals of Surgery December, 1916.

[2] Dolley, D. H.: The Cytologic Analysis of Shock. Jour. Medical Research, 1916, xxxiv.

CONCERNING THE EFFECTS OF ANESTHESIA ON THE BLOOD VOLUME AND ITS RELATION TO THE PRODUCTION OF SHOCK.*

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The subject of traumatic surgical shock has been amply studied by many investigators, and the mechanism which gives rise to its clinical manifestation is now fairly well understood. But in the experimental and clinical observation of this syn-

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drome, we find little evidence of the part played by the anesthetic in its causation.

Shock is essentially a condition of depressed vitality induced by the disturbances of circulation of such a nature that the quantity of blood which reaches the heart is insufficient for the maintenance of life. The ultimate cause of the disturbance is prolonged or excessive irritation of the central nervous system.

In view of the fact that shock has its origin in nerve irritation, the service of an anesthetic in any operative procedure would be primarily to obviate or lessen the tendency to its development; for as we know, anesthesia blocks the afferent nerve paths. However, Henderson[1] in his masterful dissertation on the subject, demonstrates how an anesthesia, when unskillfully given, may not only fail to abolish the tendency to shock, but may render the nerve centers so highly sensitive as to be directly responsible for the production of shock.

Be that as it may, most authors now recognize that the most striking pathological feature of shock is the reduction of the total quantity of blood in the body. Not only is the bulk of the blood in the venous system, but the whole volume of blood is very much diminished. It is for this reason that the clinical effects of shock have so often been likened to those of severe hemorrhage; and it is admitted that the two conditions, shock and hemorrhage, may be clinically indistinguishable. My object in touching upon these points is to bring into relation the chief factors concerned in the production of shock, with some of the striking effects of anesthesia; for anesthesia, as I will presently show, does of itself cause a prompt and considerable reduction in the blood volume.

While pursuing some work on the blood sugar changes in experimental diabetes[2] I have had occasion to observe that variations in the blood volume occur even in that brief period of anesthesia which precedes the actual operation. These changes were so constant and so striking, that subsequently in studying the effect of surgical procedures upon the blood sugar[3] I was forced to the conclusion that the hyperglycemia observed under these conditions was to some extent due to a concentration of the blood, caused by the reduction of the blood volume.

The present communication is based on animal experiments, and observations were made on some forty-one cats. A large number of the animals were studied purely for the effects of the anesthesia on the blood volume, and were permitted to recover without submitting them to surgical manipulations. Others were operated on for various purposes, and determinations of the blood volume were made at different stages of the operations. This afforded an opportunity to observe blood volume changes which occur as a result of the anesthesia alone, and those incidental to the exposure and manipulation of different abdominal organs.

The changes in the blood volume were estimated by means of the method which I have described in the Journal of Laboratory and Clinical Medicine, May, 1916[4]. This method assumes, correctly, I believe, that the total quantity of cells in the blood in one and the same individual does not vary appreciably within short periods of time; and the computation is based on the principle that a change in the relation between the fluid portion and the cell content of the blood is an indication of the change in the total blood volume. In other words, if at the beginning of an experiment the blood shows that the cells occupy 50 per cent of the total volume in a unit of blood, and one-half an hour later, from one cause or another, the cell content rises to 55 per cent of the volume, the blood has undergone concentration. The extent to which the volume of blood has become reduced under such conditions is equal to 50/55=90 per cent. Conversely, if the blood cells are found in the second examination to be only 45 per cent., the blood has become diluted, and the increase in the blood volume which can cause such dilution is 50/45=110 per cent.; so that in the first instance we have a reduction in the blood volume from 100 to 90 per cent. of the original volume; in the other we have an increase in the blood volume from 100 to 110 per cent.

Thus, without attempting to establish the exact volume of blood that is in circulation in each individual case, we can by these simple means determine the exact degree of variation that it may undergo from different influences.

By speaking of concentration of the blood, I mean concentration of the whole blood, and not merely that of the serum or plasma. Concentration or dilution of the serum or plasma may occur as a result of addition or abstraction of soluble solids, without there being any change in blood volume. This distinction is important, because reports based on changes in concentration of the blood serum do not furnish conclusive evidence that the blood volume has been altered thereby. Even changes in viscosity of the blood may lead to incomplete or erroneous interpretation. Thus Burton-Opitz[5] observes that alcohol inhalation raises the viscosity and increases the specific gravity of the blood, and attributes these changes

TABLE I.

Susceptible Animals—Operated—Ether Anesthesia.

Cat No.	Date and Hour.	Per Cent. Plasma Volume.	Per Cent. Cell Volume.	Per Cent. Relative Blood Volume.	Remarks:
5	Oct. 10, 1914				Normal Cat.
	9.43 P.M.	63.5	36.5	100.0	Before anesthesia.
	10.23 P.M.	59.1	40.9	89.2	End of operation for nephrectomy.
		63.9	36.1	101.1	Next morning.
12	Nov. 3, 1913				Normal Cat.
	4.15 P.M.	64.6	35.4	100.0	Before anesthesia.
	5.45 P.M.	57.0	43.0	82.3	End of operation for nephrectomy and pancreatectomy.
20	Dec. 19, 1914				Fasted Cat.
	4.10 P.M.	62.0	38.0	100.0	Before anesthesia.
	4.20 P.M.	58.0	42.0	90.0	During operation.
	4.45 P.M.	56.7	43.3	87.0	End of operation for pancreatectomy and nephrectomy.
	11.00 P.M.	65.2	34.8	108.0	
21	Dec. 19, 1914				Fasted Cat.
	4.55 P.M.	60.9	39.1	100.0	Before anesthesia.
	5.25 P.M.	50.0	50.0	78.2	End of operation for pancreatectomy and nephrectomy.
		64.0	36.0	108.6	16 hours later.
23	Jan. 8, 1915				Fasted Cat.
	2.40 P.M.	57.5	42.5	100.0	Before anesthesia.
	2.55 P.M.	53.0	47.0	90.4	During operation.
	4.10 P.M.	54.2	45.8	93.0	End of operation for pancreatectomy and nephrectomy.
	10.45 P.M.	59.2	40.8	104.0	6½ hours later.
24	Jan. 8, 1915				Normal Cat.
	4.25 P.M.	46.1	53.9	100.0	Before anesthesia.
	5.00 P.M.	43.8	56.2	95.0	End of operation for nephrectomy.
	10.55 P.M.	44.4	55.6	97.0	5 hours later.
	12.30 A.M.	46.1	53.9	100.0	8 hours later.
25	Jan. 9, 1915.				Fasted Cat.
	8.15 P.M.	48.7	51.3	100.0	Before operation.
	9.45 P.M.	39.5	60.5	84.0	End of operation for pancreatectomy and nephrectomy.
		51.0	49.0	104.7	Next morning.
26	Jan. 9. 1915.				Fasted Cat.
	9.55 P.M.	61.4	38.6	100.0	Before anesthesia.
	10.20 P.M.	57.0	43.0	89.8	End of operation for nephrectomy.
		61.7	38.3	100.8	Next morning.
29	Jan 25, 1915.				Normal Cat.
	8.05 P.M.	48.2	51.8	100.0	Before anesthesia.
	8.42 P.M.	43.3	56.7	91.4	End of operation for nephrectomy.
		48.4	51.6	100.5	Next day.
31	Apr. 19, 1915.				Fasted Cat.
	8.40 P.M.	55.2	44.8	100.0	Before anesthesia.
	9.40 P.M.	48.6	51.4	81.0	End of operation for pancreatectomy.
		54.1	45.9	97.9	Next morning.
40	Mar. 9. 1917.				Normal Cat.
	1.30 P.M.	57.0	43.0	100.0	Before anesthesia.
	1.45 P.M.	51.2	48.8	88.1	Abdominal incision.

to an accumulation of excretory material in the blood. Whereas this may be true on the basis of the results obtained, another conclusion is admissible,—namely, that the inhalation of alcohol causes a passage of water out of the blood into the tissues. This may cause an increase in the concentration of the serum, and also reduce the whole blood volume.

Sherrington and Copeman [6] have found that in traumatic shock the specific gravity of the blood is markedly increased, and Yandel Henderson [7] interprets these findings as evidence of a decrease in the blood volume.

Changes in the cell count or in the hemoglobin content of the blood may be regarded as better evidence of volumetric variations of the blood than changes in viscosity or specific gravity; and although these methods have been used in the study of such problems in different experimental investigations, they have not been applied to the study of blood volume in shock. Hence, the validity of the conclusions concerning the reduction of blood volume in shock might be questioned; although most authors agree that in shock the blood volume is very much reduced. Important proof that a state of

TABLE II.

Ether Anesthesia—Susceptible Animal—No Operation.

Cat No.		Per Cent. Plasma Volume.	Per Cent. Cell Volume.	Per Cent. Relative Blood Volume.	Remarks:
38	Before anesthesia	58.0	42.0	100.0	
	8 minutes later	55.0	45.0	93.3	lightly under
	21 minutes later	54.3	45.7	91.9	deeply under
	29 minutes later	58.0	42.0	100.0	reacting
	32 minutes later	60.0	40.0	105.0	no anesthesia
	36 minutes later	60.1	39.9	105.0	no anesthesia
	40 minutes later	57.4	42.6	98.6	lightly under
	47 minutes later	55.3	44.7	94.0	deeply under

This protocol shows that in the susceptible animal the blood volume becomes reduced when narcosis is induced, and again returns to normal and even increases temporarily when the anesthesia is stopped. Re-administration of the anesthesia causes again a fall in bood volume.

TABLE III.

Refractory Animals—Operated—Ether Anesthesia.

Cat No.	Date and Hour.	Per Cent. Plasma Volume.	Per Cent. Cell Volume.	Per Cent. Relative Blood Volume.	Remarks:
6	Oct. 13, 1914				Normal Cat
	8.30 P.M.	68.1	31.9	100.0	Before anesthesia
6	9.10 P.M.	70.4	29.6	102.9	End of operation for nephrectomy
8	Oct. 22, 1914				Normal Cat
	9.10 P.M.	57.9	42.1	100.0	Before anesthesia
	9.50 P.M.	60.0	40.0	105.0	End of operation for nephrectomy
32	Apr. 19, 1915				Fasted Cat
	9.45 P.M.	56.3	43.7	100.0	Before anesthesia
	10.30 P.M.	58.1	41.9	104.3	End of operation for pancreatectomy

TABLE IV.

Ether Anesthesia—Refractory Animal—No Operation.

Cat No.	Duration of anesthesia.	Per Cent. Plasma Volume.	Per Cent. Cell Volume.	Per Cent. Relative Blood Volume.	Remarks:
39	Before anesthesia	60.7	39.3	100.0	
	8 minutes later	59.9	40.1	98.0	lightly anesthetized.
	16 minutes later	62.1	37.9	104.0	incompletely anesthetized
	27 minutes later	61.4	38.6	102.0	incompletely anesthetized
	32 minutes later	60.0	40.0	98.2	incompletely anesthetized
	37 minutes later	58.0	42.0	93.0	fairly deep anesthesia
	38 minutes later	61.5	38.5	101.0	anesthesia stopped.
	45 minutes later	62.5	37.5	104.8	reacting

This protocol shows that while the animal was refractory no marked change in the blood volume occurred. During the short period of deep anesthesia, however, its blood volume did become considerably diminished.

TABLE V.

Cat No.	Source of Blood.	Per Cent. Plasma Volume.	Per Cent. Cell Volume.	Remarks:
27	Ear Vein	45.9	54.1	Animal under ether anesthesia.
	Gastric Vein	45.6	54.4	Results show the uniform distribution of cells and plasma in different parts of the circulation.
	Portal Vein	45.9	54.1	
	R't Aurice	45.8	54.2	
28	Ear Vein	42.9	57.1	Animal under ether anesthesia.
	Portal Vein	42.2	57.7	Results similar to those obtained in above experiment.
	R't Aurice	40.8	59.2	

oligemia or reduced blood volume exists in shock is furnished by the work of Mann.[8] From the experiments which he performed this observer draws the conclusion that the clinical signs of shock are due to a loss of circulatory fluid.

To summarize then, it is generally agreed that the most striking change in the blood in the course of shock is the diminution of its volume. This is not regarded as an effect of shock, but rather as one of the potent factors concerned in its develop-

ment. Nor does this mean that *"the bulk of the blood is transferred from the arterial to the venous system."* It means that the blood content of the entire circulatory system is diminished, not as a result of a change of distribution, or a loss by hemorrhage.

The reduction in blood volume is brought about by a passage of fluid from the blood to the tissues. The mechanism of this may be twofold in character. It may be, as Malcolm[9] believes, due to the spastic state of the smaller arteries and the engorgement of the venous trunks, which leads to a transudation of serum; or as Henderson[10] believes, it may be due to an altered state of the tissues brought about by an acute *acapnia,* or reduced carbon-dioxid content of the blood, which causes an increased imbibition of fluid by the tissues.

In the experiments which I have performed, two anesthetics, ether and chloroform, were used. Inasmuch as ether is the anesthetic of choice, both in clinical and experimental work, the larger number of observations were made with this drug. In the present communication the discussion will therefore be devoted to the effects of ether. The results obtained may be divided into two distinct groups: (1) Those obtained on animals which yielded readily to the anesthetic. (2) Those obtained on animals which proved refractory to the action of the anesthetic. The majority of animals tested reacted normally to the ether. In this group, Table I, and II, the blood volume is promptly affected by the anesthesia. As may be observed, the blood volume becomes reduced, and then gradually returns to normal, as the effect of the anesthesia wears off. In the second group—that is in animals which can not be rendered deeply anesthetic, the changes in blood volume are insignificant as shown in Tables III and IV.

In order to determine whether the blood volume changes bear any definite relation to the susceptibility of the animals to the anesthetic, several tests were made on cats which were at first anesthetized deeply, allowed to recover, and again rendered deeply anesthetic. The results obtained in this set of tests are exemplified by one of the protocols in Table II. These results proved that animals which respond readily to the action of the anesthetic, show a prompt reaction in the blood volume.

The question might naturally arise, whether the changes in the cell content of the blood (upon which the computation of the blood volume depends) observed after the administration of anesthesia, might not be due to an uneven distribution of the cells in the blood. It might be argued that as a result of the change in the tone of the blood vessels (spasm of the smaller arteries) the velocity of the blood might be delayed, and in consequence, circulation of the cellular elements may be impeded. This could give rise to a greater cell concentration in the blood in some parts of the circulation than in others.

To determine this point, the following experiment was performed: A specimen of blood was obtained from the ear-vein of a cat, and the cell content determined according to the method described. The animal was then thoroughly anesthetized, and another specimen of blood was obtained at the same point. Directly thereafter, a long incision was made in the abdomen, all of the viscera exposed, and specimens of blood obtained from different localities; and finally, by rapid exposure of the thoracic viscera, a blood specimen was also obtained from the heart. All the specimens were examined for the blood cell content, and the results derived, as shown by the protocol in Table V, indicate that the distribution of the cells throughout the circulation is uniform. In other words, the blood is not any more concentrated in one part of the vascular system than in another, under these conditions.

The mechanism which underlies the reduction in the blood volume during narcosis has not been determined in this study. Increased salivary secretion and sweating may be partly responsible for the loss of some fluid from the blood. Boothby and Berry,[11] have demonstrated that muscular work accompanied by sweating is associated with a marked concentration of the blood. That loss of fluid through these channels plays any very great part in the causation of the blood volume reduction seems unlikely from the fact that the animals which are refractory to anesthesia salivate very profusely, and behave in all other respects like the normal animals, but do not show any change in blood volume.

It seems probable that abstraction of fluid by the tissues from the blood plays the major part in the production of the phenomenon observed. This gains support from other associated conditions which are known to develop in anesthesia. It is now well known[12] that an acidosis develops during narcosis. It is also known that the lipoid content of the blood is increased during anesthesia. This latter phenomenon is ascribed to a mobilization of the substances from the tissues. From the work of Overton and Meyer,[13] we know that the potency of an anesthetic agent depends upon its power to dissolve fatty and lipoid material, by virtue of which action it can penetrate tissue cells. It would therefore seem that the acidosis and the mobiliza-

tion of lipoids favor development of a condition in the tissues which renders them partly hydrophilic —that is, capable of absorbing fluid. The work of Herlizka,[14] and that of Koch and Voegtlin,[15] offers additional support to this view. Whether or not an *acapnia,* as Henderson[16] suggests, plays a part in this process, can not be determined from the observations which I have made. According to this view, the animals which proved refractory to the anesthetic should have been particular predisposed to showing changes in blood volume. The fact that they fail to show any such variation seems to offer a divergence from the view that *acapnia* plays the rôle ascribed to it.

CONCLUSIONS.

This study furnishes data concerning the effect of anesthesia on the blood volume. In susceptible animals the blood volume is reduced in considerable quantity and very promptly. The difference in respect to the blood volume observed in animals which were anesthetized (and not operated) and those which were subsequently operated on, is one of degrees; for the extent of the reduction of the blood volume in the operated animals is somewhat greater than in the others. This difference can not be ascribed to a difference in duration of anesthesia, because the change in the blood volume is usually a very prompt one, and prolongation of the anesthesia seems to have little effect upon the phenomenon. On the other hand, recovery from the anesthesia is accompanied by a return of the blood volume to the normal.

In view of the fact that one of the most striking changes in shock is the reduction of blood volume, attention is therefore called to the effect of anesthesia on the blood, because it may initiate the very changes that are acknowledged to be such constant accompaniments of shock.

BIBLIOGRAPHY.

1. Henderson, Yandel; 17th Int. Cong. Med., London. 1913; Sub. & Section vii (b) Discussion 3, 49.
2. Epstein, Albert A., & Baehr, George; J. Biol. Chem. 1916; xxiv, 1.
3. Epstein, Albert A., & Aschner, Paul; J. Biol. Chem. 1916; xxv, 151.
4. Epstein, Albert A., J. Lab. & Clin. Med. 1916; i, 610.
5. Burton-Opitz, R.; Am. J. Phys. 1894; xxxv, 265.
6. Sherrington & Copeman; J. Phys. 1893; xiv, 52.
7. Henderson, Yandel; Am. J. Phys. 1910-11; xxvii, 164.
8. Mann, Frank C.; J. Gynec. & Obst. 1915; xxi, 430.
9. Malcolm, John D.; Trans. Med. Soc., London, 1909; xxxii, 274.
10. Henderson, Yandel; Am. J. Phys., 1910-11; xxvii, 161.
11. Boothby, W. M., & Berry, F. B.; Am. J. Phys., 1915; xxxvii, 378.
12. Morriss, Wm. H.; J. Am. Med. Ass., 1917; lxviii, 1391.
13. Overton & Meyer, H.; Handbuch d Path. & Pharmak, von R. Heniz, 1904, i part 1, 73.
14. Herlizka; Arch. Fisiol., 1909; xvi, 369.
15. Koch, M. & Voegtlin, C.; Bull. Hygienic Lab. 1916; No. 103, 129.
16. Henderson, Yandel; l. c. 17th Int. Cong. Med., Lon, 1913; Sub. & Section vii (b) Discussion 3, 49.

COMPARATIVE STUDY OF BLOOD PRESSURE IN ANESTHESIA.*

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A scientific view of anesthesia favors the administration of measured doses of the anesthetic agents and careful observation of the effects which result. With this view in mind, we note the amount of the drug used for producing local, spinal, or general anesthesia and the resulting change in the body functions. In this connection a study of the blood pressure is both interesting and profitable.

METHODS OF ANESTHESIA EMPLOYED.

We have administered nitrous oxid-oxygen with the intermittent flow apparatus of McKesson, which furnishes a dosage measured in percentage of the gases, with a measured percentage of rebreathing, or the constant flow apparatus of the author, which measures the flow of each gas in liters per minute.

Our method of etherization provides a constantly measured dosage of liquid ether with a constant proportion of waste. With this method the depth of anesthesia is at all times under perfect control, and a much lighter degree of anesthesia is possible than can be accomplished by the most expert anesthetist using a less exact method. This manner of measuring ether dosage has obvious practical advantages over the estimation of percentages of ether vapor by weight or by volume, or the expression of partial pressure in terms of a column of mercury. It is not affected by the confusion which exists among vapor percentages by weight and by volume and, to a greater degree, concerning vapor tension. Since there is no instrument for directly measuring vapor tension, the expression of dosage in terms of such tension is of some inetrest, but of little practical value.

THE BLOOD PRESSURE APPARATUS.

As the blood pressure apparatus for use during anesthesia is not standardized, a description of the form which we have found most practicable will be of interest. An aneroid manometer, connected by rubber tubing to a rubber cautery bulb and to the usual five by nine rubber arm band with inelastic cover, is conveniently attached by a safety-pin to the patient's pillow. The cautery bulb has practical advantages over the metallic pump used for the

* Read during the Fifth Annual Meeting of the American Association of Anesthetists, New York City, June 2, 1917.

same purpose. It is provided with a needle valve for reducing the pressure in the cuff. The arm band is wrapped closely about the patient's arm and the outer folds secured with a safety-pin. The tube connecting the cuff with the manometer is two and a half feet long.

A flat stethoscope bell is secured over the brachial bifurcation at the elbow by an arm elastic. If a Bowles stethoscope is used, the rubber disc is replaced by the diaphragm from a telephone receiver, as the porous rubber disc absorbs moisture from the skin and becomes inefficient during the course of a long operation. The stethoscope bell is connected to suitable ear pieces by three feet of rubber tubing. This apparatus is put in place before the anesthesia is commenced. The anesthetist, sitting at the head of the table, is able to take the systolic and diastolic pressures as frequently as desired without inconveniencing the surgeon or himself. Between observations the pressure in the cuff is completely released by the needle valve.

The apparatus in constant use will suffer many hard knocks and the accuracy of the dial should be frequently tested by comparison with a standard mercurial manometer. The blood pressure record, with the record of anesthetic dosage and other observations are recorded in figures on a chart, this method being more efficient than the use of a graphic chart.

Anesthetic Dosage.

While using a method resulting in a constant proportion of waste, the dosage of inhalation anesthetics can be stated as exactly as the dosage of other potent drugs. The dosage does not vary proportionally with age, body weight, or variations in the blood pressure. There is no foundation for the general supposition that anesthetic dosage is modified by previous anesthesia or by changes in the position. As in the case of other drugs, occasional instances of susceptibility to and immunity from the effects of general anesthetics are encountered, the dosage in these cases varying from as much as twice to as little as one-fourth the usual dosage. The dosage required to maintain a definite degree of anesthesia increases in proportion to the severity of the operative trauma.

Dosage in Case of Shock.

In the presence of surgical shock, the required dosage of an anesthetic is greatly diminished. It might be supposed that this diminution in dose might be due to the fall in blood pressure. A reduction in pressure in the alveolar capillaries might explain the increased potency of a constant intra-alveolar percentage of ether vapor, but as anesthetic dosage bears no fixed relation to blood pressure under other conditions, the diminished required dosage in shock probably does not depend upon lowered blood pressure.

Observations Before and During Operations.

Blood pressure observations are of value as an index to the patient's condition during the operation and also in the preliminary examination. In interpreting the preliminary observation it is important to remember that a rise of as much as 50 mm. in the systolic and pulse pressures without much change in the diastolic pressure may be due to mental excitement. This change may be encountered either at the preliminary examination or during the early stages of the anesthesia. A considerable rise in blood pressure occurs in those cases in which cyanosis is allowed to develop in the course of anesthesia. Retching or vomiting causes an immediate rise of 10 to 30 mm.

Effect of Position.

The systolic pressure increases in the lithotomy position. It is not markedly modified in the Trendelenburg position, but in those cases where the systolic pressure has been depressed in the reverse Trendelenburg position, it is immediately raised by a change to the Trendelenburg position. To watch the blood pressure during an abdominal operation on a subject in the reverse Trendelenburg position is an experience attended with the greatest interest. The steady decline in pressure corresponding to the accumulation of the blood from the peripheral circulation in the great abdominal blood reservoir, followed by the rapid rise when the position is shifted to the Trendelenburg, affords a graphic demonstration of the peripheral theory of shock.

Shock and Blood Pressure.

Patients who are skilfully anesthetized with gas-oxygen or with ether often show no great change in blood pressure even during severe operations. We have noted many cases where operations as severe as abdominal hysterectomy, salpingo-oophorectomy, or cholecystectomy, perhaps with considerable manipulation of the intestines and the introduction of gauze packing, have been done under the usual dosage of the anesthetic without a change in the systolic or diastolic pressure at any time more than 10 mm. Frequently, however, under the usual dosage, traction on the uterus or gall bladder, manipulation of the intestine, introduction of gauze packing, removal of the prostate gland, or opening the urinary bladder and the application of heat to large, denuded areas, as in breast amputations, is followed by a sharp but usually transient fall in blood pressure. When a large dose of the anesthetic is administered because of an idiosyncracy of the patient, the same condition holds true, but when a large dose is required to maintain anesthesia be-

cause of the severity of the operative trauma, a serious decline in blood pressure invariably follows.

The greatest practical value of blood pressure observations in anesthesia is in the detection of shock. Porter states that when the diastolic pressure remains for some time at from 45 to 50 mm., it will probably not recover without aid. A diastolic pressure of 50 mm. is therefore critical.

McKesson's rule is of the greatest practical value. He states that a typical case of shock is characterized by a diastolic pressure of 60 mm. or less, a pulse pressure of 20 and a pulse rate above 120. If this state is allowed to continue for half an hour the patient will not recover.

The anesthetist who is a student of blood pressure and keeps this rule in mind will rarely, if ever, record a death from surgical shock.

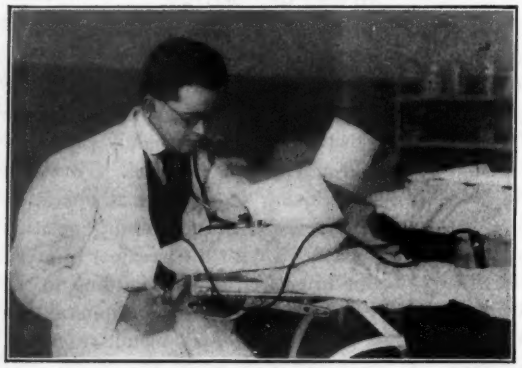

Fig. 1. Method of taking blood pressure during anesthesia.

PRESENCE OF ABDOMINAL TUMORS.

Patients with large abdominal tumors regularly present a high blood pressure, which falls rapidly on delivery of the tumor or exacuation of its contents. If, in such cases, there is some dyspnea present, a marked variation in the systolic and pulse pressures is noted during the respiratory cycle, the systolic pressure in some cases mounting 30 mm. higher in each expiration than during inspiration.

OTHER ANESTHETICS.

In the case of chloroform, Fairlie, whose observations are corroborated by Mummery, claims that the fall in blood pressure in the presence of shock is greater than under ether, but that recovery is more rapid after chloroform anesthesia.

Serious operations under local anesthesia are regularly attended with wide but transient variations in both systolic and diastolic pressure.

Spinal anesthesia is attended with a fall in systolic and diastolic pressure which is sometimes of desperate proportions.

131 WATERMAN ST.

ABSTRACT OF DISCUSSION OF THE PAPERS OF DRS. MILLER, EPSTEIN AND MUNS.

DR. S. J. MELTZER, New York City: The subject of shock is of perennial interest and yet its definition is yet to be scientifically established and its treatment clinically perfected. Undoubtedly there is something in Henderson's contention that acapnia produces shock and that rebreathing is a cure. But in the discussion of Henderson's original paper, all the physiologists did not accept his view, and I raised the point as to whether the decreased carbon dioxid content caused shock or the shock caused the decreased output of carbon dioxid. Similarly with regard to Dr. Epstein's paper, which really should be studied before being discussed, it would be difficult to determine whether loss of blood volume caused shock or shock caused the decreased blood volume.

With regard to recovery following low blood pressures, systolic or diastolic, in experiments on animals, I have found that irrespective of pressures as low as 20 systolic, if respiration could be maintained, all animals recovered. Life is, after all, dependent on the integrity of respiration and circulation, but the latter will withstand more variation than the former, and in this connection it must always be remembered that ether paralyzes the peripheral nerve centers and respiration is much less than normal during anesthesia. Animals will recover from 6, 8 or even 36 hours of anesthesia if respiration is sufficiently maintained. The control of the one factor so important to life assists in controlling the other.

DR. JAMES T. GWATHMEY, New York City: Anesthetists are generally familiar with the picture of shock—the profound relaxation, the opened eye-lids, the dilated pupil, the rapid pulse, the feeble respirations, the pallid, cold, clammy skin. Patients should never come to this crisis. In the prophylaxis of shock, in working with one of the New York surgeons, it has been our custom to use saline hypodermoclysis at the beginning of every operative procedure that will last an hour or over. This hypodermoclysis enables the surgeon to obviate the occurrence of shock and 95 per cent. of the patients so handled come off the table with a pulse of 72, normal respiration and in a most favorable condition for recovery.

With regard to the role of anesthesia in precipitating acidosis, I desire to announce some experimental results of recent investigations that will be published later in detail. Series of guinea pigs have been anesthetized with ether-air and ether-oxygen, some receiving preliminary bicarbonate of soda alkalinization. Those pigs not receiving the bicarbonate of soda were all acidosed, whereas those who received the bicarbonate of soda showed normal or raised alkalinity of the blood. Further, those pigs anesthetized with ether-oxygen showed a higher per cent. of alkalinity than those anesthetized with ether-air.

DR. E. I. MCKESSON, Toledo, Ohio: In regard to the question of cardiac compensation, which has been referred to, I have seen a number of cases in which the systolic blood pressure has been reduced, 40, 50 or 60 mm. of Hg., for a few moments and the patients recovered. But when extremely low pressures have persisted for 20 to 30 minutes, the longer they continued the less likely was the patient's recovery. These patients may not die for 2 or 3 days, but they do die. They seem unable to recover cardiac tone. When diastolic blood pressure is reduced, but the systolic pressure is maintained at practically the normal level for the patient, we have a cardiac compensation, which saves the life of the patient. The heart moves a larger volume of blood at each stroke and thus sustains systolic pressure. The double pressure maintains circulation and saves the patient's life, even though the diastolic pressure, which is the measure of the hypotonus, has indicated that the patient is approaching, but has not yet reached the stage of surgical shock.

When the heart from organic disease, severe strain, infection or rapid action has been worn out, or worn to the point where it will not compensate, we have a case when the patient is apt to go into surgical shock and not recover. It may take several days after the operation before the patient will finally succumb to heart exhaustion.

AN EVALUATION OF ETHYL CHLORID ANESTHESIA.*

MARTIN M. WARE, M. D., F. A. C. S.
NEW YORK CITY.

I gladly respond to the invitation of a most accomplished anesthetist—your secretary—to bespeak the worth of ethyl chlorid, because of my undiminished confidence in its efficacy since I first made use of this agent sixteen years ago.

The vulnerable point of any anesthetic is the mortality attendant upon its use. Being engaged in the practice of surgery, I have not followed the comparative death statistics of various anesthetics, and therefore speaking solely for myself, in the administration of many thousands of ethyl chlorid anesthesias conducted for me, I have no death to report.

In a communication reported to the Society of Surgeons of Paris but a few weeks ago, Dr. Cazin extolled highly the value and safety of ethyl chlorid, in support of which he cites the statistics of Malherbe of 6,275 cases of ethyl chlorid anesthesias, including infants a few months old and adults of every age, in which there was not a single complication. Cazin has operated with ethyl chlorid for an hour to an hour and a half with excellent results. Such has been also my experience.

An evaluation of the death rate based on an analysis of all of the statistics to hand leads me to claim a mortality of one in 15,000. I arrived at this approximate figure by eliminating such deaths coincident but not incident to ethyl chlorid anesthesia.

Thus irrelevant is the death of a child twelve months old, with diphtheretic obstruction of the larynx, when about to be tracheotomized. Another death occurred during the removal of adenoids and tonsils in a child which necessitated tracheotomy. The surgeon thinks a piece of the growth fell into the larynx. Another death is reported twenty-four hours after ethyl chlorid. Operation, gastro-enterostomy. Finally we come to a death following the discontinuation of ether which had been preceded by ethyl chlorid, and local anesthesia. In computing the death rate for ether or chloroform, we would eliminate such fatalities and assign the responsibility to contributary negligence of the anesthetist. In fact, every commentator on the topic of ethyl chlorid mortality is impressed with the fact that other elements present, independent of the anesthetic, may be responsible for the mortality.

The very fewest deaths during its administration are assigned to cardiac or respiratory failure. But this is not surprising when one takes into consideration the very brief duration of most of the ethyl chlorid anesthesias. Yet the occurrence of these fatalities is particularly heralded in the earliest stages of ether or chloroform anesthesia. It is meet at this moment to emphasize that in the very larger field of the application of ethyl chlorid at the present time, preliminary to ether, no hasty death from respiratory paralysis or cardiac failure is reported. Such assurances ought to dispel any fear as to its safety and efficiency upon longer administrations. Facts borne out by actual experience prove that its field is by no means limited to introductory anesthesia.

The safety of ethyl chlorid anesthesia introductory to ether is no longer impugned. I therefore bid fair to predict an ever-widening field of its application. Exceptionally is its use indicated for very long anesthesias. The main objection here being its expense. In many regards it outdistances nitrous oxid. It causes no cyanosis, no increase of blood pressure, no increased flow of blood and no rigidity. The quantity at hand is always visible, and no expensive or bulky apparatus is required. Attempts to widen the field of nitrous oxid and to overcome the disadvantages are familiar to us in the recommendations to precede its use with an injection of morphin and during its exhibition for long anesthesias to use oxygen. These same adjuvants, coupled with ethyl chlorid, will also greatly enhance its sphere of utility and safety.

There are a vast number of painful conditions calling for the use of the knife or some equally painful procedure which, to the opprobrium of the physician, are not given the benefits of an anesthetic. Contrast this with the practice of dentists, whose cornerstone is nitrous oxid. What explanation have we to offer for such dereliction? It is that the physician and the surgeon have not at their disposal the outfit required for administering nitrous oxid. Viewed from every angle, ethyl chlorid is the most expedient anesthetic. A dentist of this city, recently deceased, used ethyl chlorid exclusively during the eighteen years of his private practice. He enjoyed a very large practice and won renown as a skilful extractor, because, with the aid of ethyl chlorid, he could do many extractions which others did not venture under nitrous oxid. His cases, which numbered in the thousands, were unattended by any mortality. Here is a test of safety conducted under the most exacting conditions.

Not a little antagonism against ethyl chlorid arises from its improper administration. This is the crux of the matter. The same precautions are

* Read during the Fifth Annual Meeting of the American Association of Anesthetists, New York City, June 2, 1917.

called for as in the administration of the more favored anesthetics. If the ethyl chlorid is sprayed upon several folds of gauze with a very free admixture of air, a perfect anesthesia will follow somewhat more slowly, and be maintained with difficulty, owing to the volatility of the agent; and this wasteful method of using ethyl chlorid will be most expensive. The use of inhalers with rubber-bag reservoirs confines the volatilized ethyl chlorid, thereby reducing the quantity primarily used. The free admixture of air in sufficient quantity, with to and fro breathing of the bag content as with ether, favors a speedy and pleasant narcosis. When thus administered it is surprising how profoundly narcotized, completely anesthetized and thoroughly relaxed the subject becomes after a brief, preliminary and variable stage of excitement, more noticeable in neurotics and alcoholics.

This safe and efficient narcosis may be continued at pleasure and it is equally astonishing to witness a recovery to complete consciousness within a few minutes, attended by such untoward complications as nausea and vomiting in exceptionally few instances and in a diminishing degree after proper preparation of the patient. The same degree of complete narcosis attained with ether or chloroform requires hours for recovery. With nitrous oxid pure or in combination with oxygen no such relaxation is ever witnessed. The amount of ethyl chlorid and the degree of anesthesia have to be gauged by the same criteria as any other narcosis and are too elemental to be described in detail.

In conclusion, I wish to thank you for the privilege afforded to address this body of anesthetists. I regard it rather ominously good for the survival and growth of ethyl chlorid anethesia that you sought to listen to the counsels of a surgeon regarding a foremost instrumentality in the pursuit of his work.

27 EAST 81ST STREET.

ABSTRACT OF DISCUSSION.

DR. A. F. ERDMANN, Brooklyn, N. Y.: I believe Dr. Ware and I are the local godfathers of ethyl chlorid anesthesia. This (showing inhaler) is a cornucopia inhaler I first showed in 1899. Combined with the capillary stem ampoules originally manufactured for me by Friess Bros., it offers a simple, satisfactory and efficient method of administering ethyl chlorid. I use ethyl chlorid a great deal and can recall administrations lasting three-quarters of an hour. It excells as a preliminary to other anesthetics and is very valuable in combination with nitrous oxid. The latter, however, is better suited to longer operations. Rigidity does occur occasionally and the general spasm of the muscles of the body, especially the masseters, may precipitate fatality. At present the cost of ethyl chlorid is decidedly increased and militates somewhat against its use.

DR. I. D. KRUSKAL, Brooklyn, N. Y.: I have used ethyl chlorid for a number of years and have records of 7,000 administrations, in most of which it was used to induce anesthesia. The principle objection I have to ethyl chlorid is its tendency to promote nausea and vomiting, irrespective of whether it is given with abundant air supply or oxygen. Given by an open method it is the ideal anesthetic for children. It does not provide a great deal of relaxation, but I have found it serviceable in the reduction of fractures, in which considerable muscular relation is required.

DR. R. H. FERGUSON, East Orange, N. J.: I wish to call attention to the use of an apparatus employed by Dr. Guy, of the University of Edinburgh, which provides for the combined use of nitrous oxide and ethyl chlorid. One cannot witness the speedy induction, satisfactory operating and almost immediate recovery of the patients submitting to dental procedures in Guy's clinic without realizing that this combined anesthesia is a valuable method. The ethyl chlorid provides a longer operating period than the use of nitrous oxid alone. Personally, from an experience of several thousand administrations I have not always been able to secure relaxation. The routine use of this anesthetic has been opposed because the contraindications to its employment and its reactions have not been observed and studied. If administered in the presence of stenosis of the air passages or status lymphaticus it may cause trouble. Ethyl chlorid is comparatively safe in the hands of experts, but dangerous when administered by those who wish to push it to the point of securing relaxation and overcoming tonic or clonic spasm which are prone to occur during its maintenance.

DR. A. F. ERDMANN (closing): Relaxation under ethyl chlorid is usually obtainable, although with difficulty in a few instances. It is imperative to stop short of stertorous breathing in its administration. With this anesthetic available it is really a reproach to the medical profession at large that painful procedures should ever be performed without its use. Few physicians, aside from anesthetists, have the facilities for nitrous oxid anesthesia, but ethyl chlorid is available for all and the trick of using it safely can be readily acquired. Physicians should be encouraged to administer ethyl chlorid as their routine, emergency anesthetic.

THE INTERSTATE ASSOCIATION OF ANESTHETISTS WILL HOLD ITS THIRD ANNUAL MEETING IN TOLEDO, O., OCTOBER 9, 10, 11, in CONJUNCTION WITH THE MISSISSIPPI VALLEY MEDICAL ASSOCIATION. AN INTERESTING AND INSTRUCTIVE SCIENTIFIC PROGRAM HAS BEEN ARRANGED. THERE WILL BE A SPECIAL SECTION ON ANESTHESIA FOR ORAL SURGERY AND DENTISTRY. ANESTHETIC AND WAR SURGERY FILMS WILL BE SHOWN. MEMBERS OF THE ALLIED PROFESSIONS AND THEIR LADIES IN ATTENDANCE WILL BE DELIGHTFULLY ENTERTAINED.

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COL. T. H. GOODWIN, R.A.M.C., OF THE BRITISH WAR MISSION WILL ADDRESS THE JOINT-SESSION ON THE MEDICO-MILITARY PROBLEM BEHIND THE BATTLE FRONTS AND HOW THE ALLIES HAVE SOLVED THEM.

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MOVING PICTURES WILL REPLACE CLINICS AND NEW FILMS WILL BE SHOWN BY DRS. IRA O. DENMAN, JOS. BECK, C. E. TURNER AND W. I. JONES, A. M. NODINE, A. E. SMITH, RICH. H. RIETHMULLER, THOMAS ALLERS AND M. ECKER.

American Journal of Surgery

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F. HOEFFER McMECHAN, A.M., M.D., *Editor*
Avon Lake, Ohio, U.S.A.

OCTOBER EDITORIALS. 1917

THE NURSE-ANESTHETIST CONTINUED.

From an opinion of Attorney General McGhee of Ohio, recently rendered Senator Howell Wright, who represents the proponents of the nurse-anesthetist, it appears that the previous stand of the Ohio State Medical Board, declaring the nurse-anesthetist illegal, will be forcibly maintained. Senator Wright has announced his intention of taking the matter into the courts, but the law in Ohio has a clause in the section referring to registered nurses which provides that nothing in its context shall come in conflict with the existing medical practice act. The law in Kentucky did not specifically apply to nurses and consequently the Court of Appeals was forced to allow nurses to administer anesthetics and do anything else in medicine or surgery. This lack of regulating the privileges of nurses will be controlled by remedial legislation.

The Attorney General of Tennessee has also ruled against the nurse-anesthetist.

In view of the fact that Dr. A. D. Bevan, President Elect of the A. M. A., and Chairman of the Council on Medical Education, who for seventeen years has fought for higher standards in medical education and training, has definitely aligned himself in favor of qualified and licensed medical and dental anesthetists, it looks as if the matter of the nurse-anesthetist were reaching a point where it will be handled decisively.—McM.

RESEARCHES IN ANESTHESIA.

The close student of the historical evolution of anesthesia is quite aware of the fact that even ten years after the pioneer discoveries of Long, Morton, Wells, and Simpson, ether and chloroform anesthesia were so dangerous and unsatisfactory, that had not Dr. John Snow, anesthetist to the famous surgeon Liston, gone into his laboratory, where he had previously studied asphyxia, and placed anesthesia on a scientific basis of laboratory research, this beneficence to humanity might have fallen into disuse.

There is really no need to emphasize the value of research in anesthesia. The present issue of the SUPPLEMENT speaks for itself. The investigations of Davis, Muns, Epstein, Mann and others indicate clearly that all the underlying problems of anesthesia are far from solved. Their efforts to add to the science of anesthesia as well as to its art and practice is not only commendable but should prove very stimulating. If the scientific programs of the associations of anesthetists maintain the standards already set, these organizations will certainly be achieving their primary purpose. THE EDITOR will appreciate being kept in touch with the researches of other investigators.—McM.

THE NATIONAL DENTAL ASSOCIATION AND ANESTHESIA.

It will be recalled that the National Dental Association at its meeting in 1916 accorded the Interstate Association of Anesthetists the courtesy of being its Section on Anesthetics. This year, the National Dental Association has its own Committee on Anesthetics, and its secretary, Dr. Chalmers J. Lyons, of Ann Arbor, Michigan, announces the following papers for the New York City meeting, October 22-26: "The Teaching of Conductive Anesthesia," Theodore Blum, D. D. S., New York City, "After-Pain in Local and General Anesthesia," Arthur E. Hertzler, M. D., Kansas City, Mo., and "The Toxicity of Local Anesthetics," George B. Roth, M. D., Washington, D. C.

The subjects are pertinent and the essayists preeminent members of the allied professions. It is certainly pleasing to note that the meeting in Louisville cemented such cordial relations that the National Dental Association has found it desirable to continue the precedent of having medical as well as dental experts in anesthesia on its programs. This is another indication of how closely the interests of the allied professions are being consolidated.—McM.

THE CALL TO THE COLORS—SPECIAL SERVICE IN ANESTHESIA.

With the call to the Colors resounding throughout the homeland, anesthetists have been and are now responding with alacrity. Medico-military necessity as well as the continued welfare of the civilian population will make extended and tremendous demands on the medical profession at large and on the members of certain specialists in particular. Many anesthetists are already doing their bit in the war hospitals behind the far-flung battle lines of Flanders and France. Many more are required and these are promptly requesting commissions from the Surgeon General, indorsed with a plea for immediate service, which will, no doubt, be accorded them.

Major Grayson P. Murphy, heading the Red Cross Commission to France, recently cabled for a shipment of 100,000 half-pound tins of ether and for the machinery, operatives and material for establishing a large nitrous oxid-oxygen plant in France, as this form of surgical narcosis will be routinely used in the graver operations. The fact that this difficult technic will be extensively employed is evidence enough that many experts will also be required for its administration. These experts cannot be withdrawn from the hospitals at home without leaving competent and trained anesthetists to take their place. Consequently those who anticipate entering service should provide some one to do their work during their absence.

The problem involved in maintaining the supply of anesthetists is such that the interest of the National Council of Defense, the Red Cross, the Surgeon General, the allied war missions, has been sought in cooperation with the efforts of the organized anesthetists to work out an efficient plan.

Women physicians, specializing in anesthesia, will have an opportunity of serving and should immediately communicate with Dr. Alma Vedin, 224 East Fifteenth Street, New York City, who is chairman of the Subcommittee on Anesthetists of the Women's War Mission, organized under the direction of Dr. Rosalie Slaughter Morton, who has already been decorated by the French Government for her services in the war zone.

Anesthetists should make every effort to meet the grave responsibilities that are at hand. That they will do so is a foregone conclusion.—McM.

While patriotism demands sacrifices, it also provides opportunities. This is the time to have a hand in recasting the ideals of the world so that all countries, henceforth, may be more desirable to live in.

Book Reviews

A carefully selected library is essential to a thorough knowledge of the science and practice of a specialty.

Proceedings Royal Society of Medicine: Section of Anesthetics: Vol. IX, 1915-16. Edited by J. Y. W. MACALISTER. Published by LONGMANS, GREEN & Co., Paternoster Row, London, 1916.

This volume of the Proceedings continues to show the ravages of war on scientific literature. It contains the Transactions of but a single meeting of the Section on Anesthetics, which was devoted to a paper on "Observations on the Influence of Anesthetics on the Temperature of the Body," by M. S. Pembrey and F. E. Shipway with a discussion of the same. This paper is of particular importance as Shipway's contention that warmed ether-vapor is strikingly protective as a method of anesthesia in the seriously wounded and those in shock, has been borne out by the clinical results of many surgeons and anesthetists in the war hospitals behind the far-flung battle lines in France.

Encyclopedia of Foods and Beverages. Compiled by ARTEMUS WARD. Buckram bound, pages 748, copiously illustrated with Engravings and Colored Plates. ARTEMUS WARD, Publisher, 50 Union Square, New York. 1917.

This work is unique and difficult to describe in brief. It might be spoken of as the pharmacognosy of dietetics. It is eminently practical and deals exhaustively with all foods and beverages, how they are raised, prepared and marketed; how to care for them and how best to use and enjoy them.

Dietitians and dietetics both suffer from a lack of information on what foods and beverages are actually available and this lack of knowledge accounts for the limited scope of the average physician, surgeon and specialist in utilizing foods and beverages for the benefit of patients as well as their delectation.

While not making any pretense of covering all the scientific details of dietetics, the Encyclopedia is by no means superficial nor solely commercial. It is an ideal volume for the hospital superintendent and for the reference of the medical and nursing staffs.

By a polyglot and unilingual glossary, the common food names are given for French, German, Italian and Swedish. There is an equally needed glossary of "culinary and bill-of-fare items."

The colored, full-page plates are really beautiful. Beginning where the standard text-books on dietetics leave off the Encyclopedia gives a vast amount of practical and scientific information about foods, natural and artificial, which every one, who has any serious interest in dietetics, food sanitation, and economics of food will find invaluable. It will save many hours of unavailing consultation of botanies, inquiries of horticulturists and other raisers and conservers of foods, and similar attempts to learn more about foods than is given in the usual text books.

The Practical Medicine Series: General Surgery: Vol. II. Edited by ALBERT J. OCHSNER, M.D., F.R.M.S., LL.D., F.A.C.S. Cloth bound, pages 608, Illustrated with line drawings, halftones and colored plates. THE YEAR BOOK PUBLISHERS, Chicago. Series 1917.

Ochsner, who assumes the editorship of this volume of the series, pays a glowing tribute to his predecessor John B. Murphy.

The anesthetic progress detailed begins with a consideration of the legal status of the nurse-anesthetists and introduces the recent test-case in the Kentucky courts. Bazy is quoted on the effects of general and local anesthesia on the aged and Coburn elucidates the advantages of alkaloidal

adjuvants. Wilkie resurrects the single dose administration of ether, which was the method of Morton and other pioneers, and was popularized some years ago as the "ether rausch" of the Germans. Wilkie has found the method available for minor surgery. Kruskal reports the results of 1000 administrations of the essence of orange-ether sequence by the closed method, and Gwathmey volunteers to demonstrate that warmed ether with oxygen is as good, from every viewpoint, as gas-oxygen anesthesia. McCarty and Davis do not corroborate the contention that warmed ether vapor has any peculiar adantages, while Pembrey and Shipway insist that warmed anesthetics do have a beneficial effect.

The ethermoter of Montgomery is described, as are also the masks of Kingsford, Wilkie and Engstad.

Alma Vedin's experiences with endotracehehal insufflation anesthesia are given as well as W. M. Johnson's and Evropin's results with ether-oil colonic anesthesia.

The discussion of the safety of nitrous oxid-oxygen anesthesia, raised by J. F. Baldwin is supplemented by the opinions and results of Coburn, A. H. Miller, Gwathmey, McCardie, Robinson and others.

Ethyl chlorid is coming into its own in war surgery and MM. Carles and Charrier cordially indorse its use. Peck and Meltzer have used magnesium sulphate anesthesia, intravenously, in several cases, but its safety and utility are still doubtful.

Farr and Bartlett show devices for facilitating local anesthesia when used for a number of patients.

Stanley, Holman and Gadgil report their successes with spinal anesthesia, and Allen, Hertzler and Lewis their results with epidural anesthesia.

Accidents and complications of anesthesia are considered by G. Schiel, H. R. Phillips, A. Vedin and F. H. McMechan.

The remainder of the volume details the progress of General Surgery and indicates the advantages of certain methods of anesthesia or analgesia for specific operations, when necessary.

Society Proceedings

Membership in a society devoted to your specialty is an essential to success.

INTERSTATE ASSOCIATION OF ANESTHETISTS THIRD ANNUAL MEETING—TOLEDO, O., OCTOBER 9, 10 AND 11.

Beside the papers of the scientific program already announced for the Third Annual Meeting of the Interstate Association of Anesthetists, Hotel Secor, Toledo, O., October 9, 10 and 11, in conjunction with the Mississippi Valley Medical Association, the following features have been arranged for.

Moving picture demonstrations will be given in the Temple Theatre by Dr. M. Ecker, Low Pressure Nitrous Oxid-Oxygen Anesthesia by Nasal Administration; Dr. Thomas Aller, Extractions under Nitrous Oxid-Oxygen Anesthesia; Dr. Ira O. Denman, Operations in the Forward Inclined Sitting Posture; Dr. Joseph C. Beck, Vapor Anesthesia for Oral Surgery; Dr. A. E. Smith, Methods of Conductive Anesthesia; Dr. Rich. H. Riethmuller, Why Conductive Anesthesia Fails; Dr. Pont, Plastic Surgery of the Face and Jaw. Other films depicting recent advances in war surgery and showing the work of the Red Cross will be exhibited.

The speakers at the Annual Dinner will be Dr. F. G. DuBose, Selma, Ala.; Dr. George F. Butler, Mudlavia, Ind.; Dr. Otto U. King, Huntington, Indiana and Dr. Lafayette L. Barber, Toledo, O.

The Committee on Arrangements, headed by Dr. W. J. Stone of Toledo, O., has scheduled the following entertainment. October 10, Buffet Luncheon for members and ladies at the Hotel Secor. Automobile ride for the visiting ladies with tea at the Country Club. Evening: President's reception, buffet lunch and dance at the Toledo Club. October 11: Theatre party at Keith's Theatre.

Covers for the Annual Dinner will be $2.00 a person. All those contemplating attending are cordially invited to bring the ladies to lend grace and charm to the occasion.

For additional information, dinner reservations, applications for membership, address

F. H. McMECHAN, M.D., Sec'y.,
Avon Lake, Ohio.

AMERICAN ASSOCIATION OF ANESTHETISTS, FIFTH ANNUAL MEETING.

The Fifth Annual Meeting of the American Association of Anesthetists was called to order in the Blue Room of the Hotel McAlpin, at 9.15 A. M., Saturday, June 2, 1917, by Dr. F. W. Nagle, Vice-President, in the absence of President Captain Dr. Walter M. Boothby, who was unavoidably absent on account of service with the Harvard Unit at the front.

The minutes of the Detroit meeting were approved as read and the Treasurer's report accepted.

The following new members were elected: Morris H. Clark, Kansas City, Mo.; Fred W. Culler, New York City; Wm. W. Dickey, New York City; Wm. G. Doran, New York City; James W. George, Minneapolis, Minn.; Wm. M. Hall, Penneville, Ind.; Thomas R. Hanley, Toronto, Ontario; R. F. Hastreiter, Los Angeles, Cal.; R. Franklin Hill, Philadelphia, Pa.; Robert E. Jameson, Davenport, Ia.; Marie B. Kast, Indianapolis, Ind.; H. Clifton Luke, New York City; Jean M. Martin, San Francisco, Cal.; Louis H. Maxson, Seattle, Wash.; George R. McAuliffe, Chicago, Ill.; Ben Morgan, Chicago, Ill.; Anna M. Mosgrove, San Francisco, Cal.; Louise A. Oldenburg, San Francisco, Cal.; Allen Galpin Rice, Springfield, Mass.; Louise Rosenzweig, Staten Island, N. Y.; Eleanor Seymour, Los Angeles, Cal.; Everett A. Tyler, Philadelphia, Pa.; Michael Vinciguerra, Elizabeth, N. J.; R. M. Waters, Sioux City, Ia.; Charles J. Wells, Syracuse, N. Y.

In view of the Call to the Colors, the Chairman appointed the following committee, Drs. A. H. Miller, C. H. Gallegher, F. L. Richardson and Charles K. Teter, and its Resolution, as follows, was unanimously adopted:

"The American Association of Anesthetists, a body of physicians whose object is the advancement of the science and art of anesthesia, anxious to serve in the present national crisis, offers its moral support and services to the Government in the interests of efficient anesthesia in the medical service of the Army and Navy; especially in the instruction of those who are to have charge of the administration of anesthetics in active service."

Dr. Isabella C. Herb, of the Committee appointed at the Detroit meeting to investigate the teaching of anesthesia in the medical schools and teaching hospitals, reported that what material the Committee had been able to develop would be reported in the paper on: Anesthesia in the Curriculum and Clinic, by Dr. W. D. Gatch, to be read during the Joint-Session of the Sections on Physiology & Pathology and General & Abdominal Surgery.

As the papers of the Scientific Sessions will be published in the SUPPLEMENT, there is no need to refer to them in this report.

The following officers were elected for 1917-1918: President, F. W. Nagle, Montreal, Canada; Vice-Presidents, Albert H. Miller, Providence, R. I.; Isabella C. Herb, Chicago, Ill.; Freeman Allen, Boston, Mass.; Secretary-Treasurer, James T. Gwathmey, New York City; Members of the Executive Committee, Yandell Henderson, New Haven, Conn., and Charles Baskerville, New York City.

The New York Society of Anesthetists acted as host to the American Association of Anesthetists for the delightful banquet served in the evening.

Following a delicious menu, the banqueters were entertained by several noted after-dinner speakers: Dr. Wm. H. Cantle, "A Few Bubbles"; Wm. J. Burns, "The Detec-

tive and the Doctor"; Hon. Stanley H. Howe, "Charity, Its Use and Abuse"; and Wm. J. Luckett, "The Surgeon and the Anesthetist."

Through the efforts of Dr. Isabella C. Herb of Chicago, the Joint-Session of the A. M. A. Sections on Physiology & Pathology and General & Abdominal Surgery was made of especial interest to the visiting anesthetists on account of the following pertinent papers: "Shock," Frank C. Mann, Mayo Clinic; "Anethesia in the Curriculum and Clinic," Willis D. Gatch, Indianapolis, Ind.; "Pulmonary Changes Following Intratracheal Anesthesia for Experimental Lung Surgery," Conrad Georg, Jr., Ann Arbor, Mich.; and "Some Observation on the Toxicity of the Common Anesthetics," Evarts A. Graham, Mason City, Iowa.

During the A. M. A. clinics, June 4-5, the anesthetists in attendance at the New York meeting had the stimulating experience of seeing all the routine and many of the newer methods of anesthesia and analgesia demonstrated.

Quarterly Index

Of the Current Literature of Anesthesia and Analgesia.

ACIDOSIS, RELATION OF TO ANESTHESIA. G. A. Caldwell and M. Cleveland, New York City. Surgery, Gynecology and Obstetrics, July, 1917.

ALBUMINURIA, POSTOPERATIVE. A Satire. Paris Medical, May 26, 1917.

ANESTHESIA, CASE OF SAFETY PIN IN ESOPHAGUS, DISPLACED DURING, AND LODGING IN POSTNASAL SPACE. A S. Kaufman, Philadelphia. Laryngoscope, June, 1917.

ANESTHESIA IN THE CURRICULUM AND CLINIC. Willis D. Gatch, Indianapolis. Journal American Medical Association, August 4, 1917.

ANESTHESIA AND ANALGESIA IN OBSTETRICS. Harold Heffron, Matamora, O. Ohio Medical Journal, July, 1917.

ANESTHESIA AND OPERATION, EFFECTS ON KIDNEY. R. Colp, New York City. American Journal Medical Sciences, June 1917.

ANESTHESIA, SAFETY FIRST IN. T. L. Dagg, Chicago. Illinois Medical Journal, June 1917.

ANESTHETICS AT THE FRONT, ADMINISTRATION OF. G. Marshall. British Medical Journal, June 2, 1917.

ANESTHETICS, SOME ACTIONS OF, ANALYZED BY OBSERVATION OF ALTERED CARDIAC REACTION TO CALCIUM. W. Burridge. Quarterly Journal Medicine, Oxford. April, 1917.

APPARATUS-VALVE TO REGULATE DELIVERY OF AIR AND ETHER VAPOR IN ANY PROPORTIONS. F. L. Gates, New York City. Journal Experimental Medicine, July, 1917.

CHLOROFORM ANESTHESIA, FATAL JAUNDICE AND ALBUMINURIA FOLLOWING THIRD ADMINISTRATION OF. N. Feissinger and R. Montaz. Revue de Chirurgie, September-October, 1916. (Published March, 1917) xxxv, Nos. 9-10.

CHLOROFORM ANESTHESIA, PERIPHERAL PARALYSIS FOLLOWING. C. Pastine. Riforma Medica, March 31, xxxiii, No. 13, 1917.

CHLOROFORM, SAMUEL GUTHRIE, DISCOVERER OF. W. V. Ewers, Rochester, N. Y. Buffalo Medical Journal, May-June, 1917.

CONDUCTIVE ANESTHESIA FOR THE GENERAL DENTAL PRACTITIONER. F. W. Rounds, Louisville, Ky. Dental Summary, May, 1917.

CONDUCTIVE ANESTHESIA WITH NOVOCAIN FOR EXODONTIA. L. Biddle Duffield, Philadelphia. Dental Cosmos, July, 1917.

ETHER AS AN ANESTHETIC IN THE EXTRACTION OF TEETH, NOTES ON THE USE OF. Leslie Curnock, London. Dental Digest, July, 1917.

ETHER ANESTHESIA, THE TREATMENT OF TUBERCULOSIS BY. Walter E. Savage, Cincinnati, O. Ohio Medical Journal, July, 1917.

ETHYL CHLORID, GENERAL ANESTHESIA WITH IN WAR SURGERY. M. Boureau. Presse Medicale, May 17, 1917.

LEGAL ASPECTS OF GENERAL ANESTHESIA IN DENTISTRY: THE DENTIST BEFORE THE LAW. Henry Schwamm, New York City. Dental Cosmos, June, 1917.

LOCAL ANESTHESIA. F. G. Dyas, Chicago. Illinois Medical Journal, May, 1917.

LOCAL ANESTHESIA, LIMITATIONS AND ADVANTAGES OF. Harvey F. Smith, Harrisburg, Pa. Pennsylvania Medical Journal, May, 1917.

LOCAL ANESTHESIA, ABDOMINAL SURGERY UNDER. R. E. Farr, Minneapolis. Journal-Lancet, May 15, 1917.

LOCAL ANESTHESIA—THE TECHNIC OF SUPRAPUBIC PROSTATECTOMY. I. S. Knoll, Chicago. Urologic and Cutaneous Review, May, 1917.

LOCAL ANESTHESIA FOR TONSILLECTOMY. J. A. Thompson, Cincinnati, O. Ohio Medical Journal, July, 1917.

LOCAL ANESTHESIA FOR TONSILLECTOMY, SIMPLIFIED TECHNIC FOR. W. T. Patton, New Orleans. Journal American Medical Association, July 7, 1917.

LOCAL ANESTHESIA FOR HEMORRHOIDECTOMY: TECHNIC OF INFILTRATION, EXTRADURAL AND PARASACRAL ANESTHESIA: CLAMP AND CAUTERY REMOVAL. V. C. David, Chicago. Surgical Clinics of Chicago, June, 1917.

LOCAL ANESTHESIA FOR DENTISTRY. Arthur E. Smith, Chicago. Dental Items of Interest, June, 1917.

LONG, CRAWFORD WILLIAMSON AND ETHER. E. M. Magruder, Charlottesville, Va. International Journal of Surgery, July, 1917.

MONOPLEGIA, POSTOPERATIVE BRACHIAL. N. Frederici. Riforma Medica, April 21, 1917.

NITROUS OXID-OXYGEN ANALGESIA AND ANESTHESIA IN OBSTETRICS. G. G. Copeland, Toronto. Canadian Medical Association Journal, May, 1917.

NITROUS OXID ANALGESIA, USE OF IN OBSTETRICS, WITH DESCRIPTION OF A SIMPLE APPARATUS. T. H. Cherry, New York Medical Journal, June 30, 1917.

NITROUS OXID GAS A. AN ANALGESIC IN 135 CASES OF LABOR. G. H. Ryder, New York City. American Journal of Obstetrics, June, 1917.

NITROUS OXID AND ETHER ANESTHESIA, AN EXPERIMENTAL RESEARCH INTO THE NATURE OF. Geo. W. Crile, Cleveland, O. Dental Summary, June, 1917.

NOVOCAIN-SUPRARENIN. Emil Specht, New York City. Dental Items of Interest, June, 1917.

OXYGEN, COMPRESSED, FOR MEDICAL PURPOSES. Tilden Adamson, New York City. Journal American Medical Association, June 2, 1917.

RECTAL ANESTHESIA: CASE OF CESAREAN SECTION UNDER. A. E. Panting. Medical Journal of Australia, May 12, 1917.

SHOCK, SURGICAL, SUGGESTION FOR TREATMENT OF: USE OF PITUITRIN. Editorial. Journal American Medical Association, July 21, 1917.

RESUSCITATION, HISTORY AND ANALYSIS OF METHODS OF. S. J. Meltzer, New York City. Medical Record, July 7, 1917.

SHOCK DURING GENERAL ANESTHESIA. F. C. Mann, Rochester, Minn. Journal American Medical Association, August 4, 1917.

SHOCK, SURGICAL, RESPIRATORY SUCTION AS AN AID IN. W. T. Porter, Boston. Boston Medical and Surgical Journal, May 10, 1917.

SHOCK, TRAUMATIC, THE RELATION OF LOW BLOOD PRESSURE IN A FATAL TERMINATION. F. H. Pike and Helen C. Coombs, New York City. Journal American Medical Association, June 23, 1917.

STOMACH, ACUTE DILITATION OF: REPORT OF SIX CASES, THREE UNDER GENERAL ANESTHESIA. F. B. Reardon, California State Medical Journal, July, 1917.

SPINAL ANALGESIA. A. W. B. Livesay. Journal Royal Naval Medical Service, January, 1917.

SPINAL ANESTHESIA. C. C. Yount, Panama City. Surgery, Gynecology and Obstetrics, July, 1917.

SPINAL ANESTHESIA WITH TROPOCOCAIN. H. A. Cheng. National Medical Journal of China. March, 1917.